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REVIEWS OF THE ENVIRONMENTAL EFFECTS OF POLLUTANTS:

XIII. Endrin

MASTER

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REVIEWS OF THE ENVIRONMENTAL EFECTS OF POLLUTANTS: XIII. ENDRIN

Report prepared by

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FOREWORD

A vast amount of published material is accumulating as numerous research investigations are conducted to develop a data base on the adverse effects of environmental pollution. As this information is amassed, it becomes continually more critical to focus on pertinent, well-designed studies. Research data must be summarized and interpreted in order to adequately evaluate the potential hazards of these substances to ecosystems and ultimately to public health. The Reviews of the Environmental Effects of Pollutants (REEPs) series represents an extensive compilation of relevant research and forms an up-to-date compendium of the environmental effect data on selected pollutants.

Reviews of the Environmental Effects of Pollutants: XIII. Endrin includes information on chemical and physical properties; pertinent analytical techniques; transport processes to the environment and subsequent distribution and deposition; impact on microorganisms, plants, and wildlife; toxicologic data in experimental animals including metabolism, toxicity, mutagenicity, teratogenicity, and carcinogenicity; and an assessment of its health effects in man. The large volume of factual information presented in this document is summarized and interpreted in the final chapter, "Environmental Assessment," which presents an overall evaluation of the potential hazard resulting from present concentrations of endrin in the environment. This final chapter represents a major contribution by Frederick Oehme from Kansas State University.

The REEPs are intended to serve various technical and administrative personnel within the Agency in the decision-making processes, i.e., in the development of criteria documents and environmental standards, and for other regulatory actions. The breadth of these documents makes them a useful resource for public health personnel, environmental specialists, and control officers. Upon request these documents will be made available to any interested individuals or firms, both in and out of the government. Depending on the supply, the document can be obtained directly by writing to:

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ABSTRACT

This study is a comprehensive, multidisciplinary review of the health and environmental effects of endrin (1,2,3,4,10,10-hexachloro-6,7-epoxy-1,4,4a,5,6,7,8,8a-octahydro-1,4-endo,endo-5,8-dimethanonapthalene). More than 600 references are cited.

Endrin is used chiefly as an insecticide, particularly for the control of lepidopterous larvae that infest cotton crops. In 1971, total U.S. consumption of endrin exceeded 600,000 kg. Of this amount, 75% was applied to cotton crops, and 99% of the total usage occurred the southeastern and delta states.

Endrin is highly toxic to mammals, birds, fish, and insects, but is generally nontoxic to plants. Acute exposure of mammals to endrin typically results in central nervous disorders, with convulsions leading to death through respiratory failure within two days. Symptoms of acute endrin exposure in humans include convulsions, vomiting, abdominal pain, nausea, dizziness, and headaches. Chronic exposure of mammals to endrin may result in damage to the liver, kidney, heart, brain, lung, adrenal gland, and spleen. Behavorial abnormalities, reproductive disorders, changes in carbohydrate metabolism, changes in blood composition, and other effects secondary to central nervous system disorders have also been observed following chronic exposure of mammals to sublethal doses of endrin.

No malignancies attributable to endrin have been reported, but chromosomal abnormalities and teratogenesis have been induced in several mammalian species by endrin.

Endrin is dissipated from the environment by photochemical and thermal decomposition and by microbial degradation. Relatively few bacteria can degrade endrin, but many species of algae and fungi exhibit this ability. The major product of endrin decomposition under natural conditions is ketoendrin.

Environmental contamination by endrin appears to be restricted to areas where the compound is used intensively; background concentrations in the atmosphere, hydrosphere, and lithosphere at locations distant from areas of heavy use are generally below the levels of detection.

1. EXECUTIVE SUMMARY

1.1 MAJOR FINDINGS

Endrin (1,2,3,4,10,10-hexachloro-6,7-epoxy-1,4,4a,5,6,7,8,8a-octahydro-1,4-endo, endo-5,8-dimethanonapthalene), the common name for one member of the cyclodiene group of insecticides, is a cyclic hydrocarbon having a chlorine-substituted methano bridge structure (Sect. 2.2). Endrin was introduced in the United States in 1951 and is manufactured domestically by the Velsicol Chemical Corporation and in the Netherlands by Shell Nederland Chemie. The endrin sold in the United States is a technical-grade product containing not less than 95% active ingredient. It is available in a variety of diluted formulations (Sect. 7.2).

The only known uses of endrin are as an avicide, rodenticide, and insecticide, the last of these being the most prevalent. The largest single use of endrin is for the control of lepidopterous larvae attacking cotton crops in the southeastern and delta states. In 1971 (the last year for which data are available), 75% of all endrin used (total usage 6.438×10^5 kg) was applied to cotton, while 99% of the total usage was confined to the southeastern and delta states (Sect. 7.5.2).

A variety of analytical techniques are available for the qualitative and quantitative determination of endrin in environmental samples. Included among these are biological assays, colorimetric methods, thin-layer chromatography, determination of total chlorine, and infrared spectroscopy (Sect. 2.5). The method of choice, however, is gas-liquid chromatography (GLC) using an electron-capture detector. GLC methods are rapid, simple, and highly sensitive, permitting detection of nanogram quantities of endrin residues. Unequivocal confirmation may be achieved by mass spectrometry. Chemical ionization mass spectrometry of endrin results in a less complex spectrum than does the electron impact counterpart and is therefore useful for the identification of endrin in crude extracts (Sect. 2.5).

Endrin enters the environment primarily as a result of direct applications to soil and crops. Discharge of waste materials from endrin manufacturing and formulating plants and disposal of empty containers also contribute significantly to observed residue levels (Sects. 7.4.2.3 and 8.2). Environmental contamination appears, for the most part, to be restricted to those areas where endrin is used extensively. Concentrations as high as 0.64 ppm were observed in Mississippi soils during 1972, while maximum concentrations in Arkansas and Louisiana soils during 1973 were 0.24 and 0.48 ppm respectively (Sect. 7.5.3).

Monitoring of the surface waters of United States river basins between 1957 and 1966 revealed consistent contamination by endrin only in the lower Mississippi basin. In addition, the highest concentrations recorded during the six consecutive years from 1960 to 1965 were all from this area. While residue levels reported between 1970 and 1975 were significant (20 ppt), they did not approach the maximum of 214 ppt present in 1963, when numerous endrin-related fish kills occurred (Sect. 7.4.3). The major source of endrin in rivers and other freshwater bodies is surface runoff from fields and crops following application (Sect. 7.4.2).

The large-scale use of endrin in the agricultural lands surrounding the Mississippi River basin prior to 1964 was reflected in the levels of the pesticide detected in bottom deposits (10,000 ppt) in that year. The highest concentration of endrin in the sediment of any river basin between 1970 and 1975 (6700 ppt) was also found in the lower Mississippi (Sect. 7.4.3).

Extensive monitoring for endrin in the atmosphere has not been conducted. The highest concentration reported to date was 58.5 ng/m^3 in Stoneville, Mississippi, in 1971 (Sect. 7.3.3). Vaporization of endrin from treated soils and crops is believed to represent the major source of atmospheric contamination.

Background concentrations in the atmosphere, hydrosphere, and lithosphere, far removed from agricultural areas where endrin is applied and industrial areas where endrin is manufactured, are generally below the limits of detection (Sects. 7.3.3, 7.4.3, 7.5.3). However, the structural, chemical, and toxicological similarities between endrin and related compounds which have been observed at great distances from their points of application suggest that transport of endrin to remote areas is not unlikely (Sect. 7). Transport to remote areas and to the open ocean is expected to occur primarily via the atmosphere (Sect. 7.3).

Most established mechanisms for removal of endrin from the atmosphere, hydrosphere, or lithosphere do not remove it from the environment, but rather relocate it to other environmental compartments (Sects. 7.3.4, 7.4.4, 7.5.4). Included among those mechanisms which result in actual dissipation of endrin from the environment are (1) microbial degradation (Sect. 3.2.1), (2) thermal decomposition (Sect. 2.4), and (3) photodecomposition (Sect. 2.4). The major product of these reactions is ketoendrin, which, while more persistent than endrin itself, is significantly less toxic to mammals (Sect. 6.2.3).

Relatively few bacteria (2 to 17%) isolated from soil are capable of degrading endrin. When degradation does occur, ketoendrin is the predominant metabolite, although other products, including endrin aldehyde and endrin alcohol, have also been detected. Anaerobic conditions generally favor microbial degradation; bacterial transformation increases with increasing depth of field soil and when soils are flooded (Sect. 3.2.1).

Toxicity of endrin to bacteria manifests as growth inhibition. However, endrin does not appear to be highly toxic to most species of bacteria. A correlation between bacterial sensitivity to endrin and gram response appears to exist. Gram-negative and gram-variable bacteria grown on solid medium were not affected by surface films of 0.5 mg of endrin per milliliter; but 50% of gram-positive cultures experienced growth inhibition under identical conditions (Sect. 3.2.2).

Endrin is degraded to ketoendrin and other metabolites by many species of algae and fungi as well (Sects. 3.3 and 4.2). The extent to which degradation occurs varies considerably among the different organisms. Over 70% of radiocarbon-labeled endrin was recovered unchanged from cultures of *Aspergillus niger*, while an unidentified yeast culture metabolized 80.6% of the added endrin to ketoendrin (Sect. 4.2). Blue-green algae bioconcentrate endrin manyfold; however, the degree of bioconcentration appears to increase with decreasing exposure levels. A bioconcentration factor of 4500 was observed in *Oedogonium* spp. following exposure to only 2.5 ppb.

Both endrin and ketoendrin were found to inhibit growth in the blue-green alga *Anacystis nidulans* at concentrations as low as 10 ppb. Growth inhibition was also observed in two species of nitrogen-fixing blue-green algae. Inhibition was observed to increase with increasing endrin concentration (Sect. 3.3). Reduction in radiocarbon uptake and inhibition of cell division in marine phytoplankton occur following exposure to endrin. Both effects were observed in a particularly sensitive species, *Cyclotella nana*, at concentrations as low as 0.01 ppb (Sect. 3.3).

Vascular plants absorb, translocate, and metabolize endrin (Sect. 4.3.1). Absorption through the roots occurs in both root crops and other types of plants. The quantity of endrin absorbed depends upon endrin concentration and soil type and varies considerably with plant species. Carrots usually absorb more endrin than do other root crops.

The detection of endrin residues in aerial plant parts constitutes evidence for translocation following root uptake from endrin-containing soil. Foliar absorption of endrin and its subsequent translocation within the plant have also been demonstrated. Following application of ^{14}C-labeled endrin to the leaves of cotton plants, 20% of the recovered radioactivity was found in the stalks, pods, fibers, and soil, with traces observed in the roots and seeds.

As many as five different metabolites have been recovered from plants, the major metabolite consisting of an endrin alcohol. Ketoendrin, however, is the only degradation product which has been conclusively identified (Sect. 4.3.1).

Endrin is generally nontoxic to plants; there are exceptions, however. The ability of barley (*Hordeum vulgare*) seeds to germinate is reduced by exposure to endrin, the extent of reduction being a function of both endrin concentration and duration of exposure. Cytological studies of the root-tip cells of the resultant seedlings revealed chromosomal aberrations, although obvious morphological effects were not apparent in the roots themselves. Chromosomal abnormalities were transmitted to the C-2 generation following exposure of seeds to 1000 ppm endrin for 12 hr. A threefold increase over the spontaneous mutation rate resulted. Meiotic cells of seedlings sprayed with a 500-ppm solution of endrin exhibited chromosomal abnormalities at a frequency of 0.98% (Sect. 4.3.2).

The uptake of soil constituents by plants is affected by endrin contamination of the soil. Abnormalities in uptake of calcium and iron have been observed in several species. In addition, the quantities of certain plant constituents are altered when plants are grown in endrin-containing soil. Variations in amino acid composition as well as in the levels of both macro- and microelements were observed in the aerial portions of corn (*Zea mays*) (Sect. 4.3.2).

Aquatic mollusks and arthropods are exposed to endrin in the water, sediment, and food sources. These invertebrates extract endrin from their surroundings, continually exchanging residues between water and body fluids until an equilibrium is attained. The equilibrium concentration will depend upon both the concentration of endrin in the water and the species involved. Bioaccumulation factors as high as 49,000 have been observed in snails (*Physa* sp.), with tissue concentration reaching 492 ppm. Approximately 17% of ^{14}C-labeled endrin was recovered from snails in the form of metabolites, indicating that biodegradation does occur to some extent. Flushing with uncontaminated water results in rapid elimination of endrin residues from several species of aquatic invertebrates (Sect. 5.2.1.1).

Endrin is more toxic to aquatic arthropods than to mollusks. Clams (*Mercenaria mercenaria*) and mud snails (*Nessa obsoleta*) survive at levels 1000 times greater than concentrations (less than 10 ppb) lethal to several species of marine shrimp. In some species, small changes in concentration can make the difference between no effect and lethality. Effective lethal concentrations depend upon factors such as temperature, duration of exposure, and stage of development.

Other adverse effects noted in aquatic invertebrates include inhibition of shell growth, abnormalities of the nervous system, and reproductive dysfunction (Sect. 5.2.1.2).

Fish are exposed to endrin through the same mechanisms as are aquatic invertebrates. The primary means of entry into the fish body is absorption through gill surfaces, and uptake from water far exceeds that from food. For example, in channel catfish (*Ictalurus punctatus*), bioconcentration from water is 2000 times higher than from food. Endrin is rapidly eliminated from fish when exposure to contaminated water or food is terminated.

Mosquito fish (*Gambusia affinis*) biodegrade approximately 25% of absorbed endrin, and one metabolite has been tentatively identified as 9-hydroxy endrin. No evidence for biotransformation is available for other species of fish (Sect. 5.2.2).

Endrin is the most toxic to fish of the common organochlorine insecticides. The concentration lethal to 50% of experimental fish

(LC_{50}) following 96 hr of exposure is below 1 ppb in many species; 24-hr LC_{50} values of less than 1 ppb are not uncommon. The margin of safety for lethality in fish exposed to endrin is also exceedingly narrow. Spot (*Leiostomus xanthurus*) flourish at 0.05 ppb, but a concentration of 0.1 ppb causes 100% mortality in five days. The primary effect of endrin in fish is on the nervous system. The brains and nervous tissue of endrin-resistant mosquito fish absorb three to four times less endrin than do those of endrin-sensitive fish. Cell membrane fractions from brains of endrin-resistant fish bind twice as much endrin as those from susceptible fish, thus reducing the amount of endrin entering the cell. The increased binding of endrin to myelin tissue in the brain observed in fish which succumb to endrin toxicity probably results in interference with nerve transmission. Other effects on fish include reproductive abnormalities, decreased growth, impaired liver function, pathological changes in the gills and pancreas, enzyme inhibition, and increased whole-body lipid.

Birds are exposed to endrin primarily through ingestion of contaminated aquatic organisms and water. Endrin is absorbed by the gut and transported to various body tissues where accumulation occurs. Adipose tissue contains the highest levels of endrin, while brain tissue contains the lowest. Accumulation of endrin is proportional to the quantity ingested. Endrin ingested by laying hens is also transmitted to their eggs, and a direct relationship between dosage and accumulation exists.

No evidence for biotransformation of endrin by avians has been found (Sect. 5.3.1).

Lethality is the most frequently observed toxic effect in birds. Of 89 chemicals tested, endrin proved most toxic to all of four avian species studied (bobwhites, mallards, pheasants, and Japanese quail). Concentrations of endrin in feed ranging from 14 to 22 ppm produced 50% mortality in each species after eight days (five days of exposure to contaminated feed followed by three days of noncontaminated feed). The lowest daily oral dose for mallards which produced one or two deaths in 30 days was 0.125 ppm. Juvenile birds appear more susceptible to the toxicity of endrin than do adults; males succumb more readily than do females (Sect. 5.3.2).

Endrin appears to affect the central nervous system prior to death. Symptoms such as nervousness, tremors, muscular incoordination, and convulsions have been observed in a variety of species (Sect. 5.3.2).

Effects on reproduction have also been observed. Egg production, hatchability, and viability of chicks were significantly reduced in pheasants receiving 10 ppm endrin in their feed during the reproductive period. Reduced chick viability was also observed in quail following ingestion of feed containing 1.0 ppm endrin (Sect. 5.3.2).

The metabolism, distribution, storage, and excretion of endrin have been studied extensively in only one type of insect, the larvae of tobacco budworm (*Heliothis virescens fabrieius*). Following topical application of radioactive endrin, the distribution and extent of accumulation in various tissues were identical in both endrin-resistant and -sensitive strains. The rate of cuticular penetration was, however, greater in sensitive strains. Both strains metabolized endrin to ketoendrin and endrin aldehyde, but two additional metabolites were also produced by the resistant larvae. Most of the radioactivity excreted by both strains consisted of unchanged endrin (Sect. 5.4.1).

The basis of endrin toxicity to insects has not yet been experimentally determined; however, it is known that endrin affects the central nervous system, and the ganglia are believed to be the site of action (Sect. 5.4.2).

Sublethal concentrations of endrin were not mutagenic in the fruit fly (*Drosophila melanogaster*), nor did they affect reproduction or survival of progeny in lady beetles (*Colcomegillar maculata De Geer*) and red cotton bugs (*Dysdercus koenigii*). The only other sublethal effect observed has been inhibition of acid and alkaline phosphatases in the coccid (*Dactylopus confusas*) (Sect. 5.4.2).

Wild and domestic mammals are exposed to endrin primarily through ingestion of treated foliage, although dermal contact and inhalation also occur. Endrin shows little tendency to accumulate in tissues other than adipose tissue; levels ranging from 0.01 ppm (in cows) to 23.7 ppm (in lambs) have been detected in both internal and external fat of a variety of species following ingestion of endrin-fortified food. Endrin residues have been detected in milk from endrin-fed cows under natural as well as experimental feeding conditions. Levels found were proportional to both quantity and duration of endrin ingestion (Sects. 5.5.1 and 5.5.2).

Metabolism of endrin has been studied extensively in the rat (Sect. 6.2.3). Endrin is rapidly metabolized in the liver and excreted as hydrophilic metabolites. The biological half-life in male rats receiving 0.4 ppm in the diet is two to three days. In female rats, which metabolize endrin more slowly, the half-life is four days. In rats, both endrin and its metabolites are excreted primarily in the feces; in rabbits, excretion is primarily via the urine.

Endrin is highly toxic to all mammals regardless of route of exposure (Sect. 6.3.2). However, it is also rapidly eliminated, so the return to normal among those that survive is also rapid. The primary effect of acute exposure is on the central nervous system. When lethal concentrations are administered, convulsions may occur as soon as 30 min after exposure; convulsions can culminate in death through respiratory failure in about 48 hr. The dose lethal to 50% of experimental animals ranges from 0.3 mg/kg for the monkey to 50 mg/kg for the goat (Sects. 6.3.2 and 5.5.1).

Many instances of mammalian fatalities have been reported outside the laboratory. Field application of endrin at rates of 0.55 to 2.75 kg/ha resulted in the death of 33 to 100% of various species of wild mice. Forty-two percent of a herd of cows accidentally died after being sprayed for flies with a 0.34% solution of endrin (Sects. 5.5.1 and 5.5.2).

The chronic toxicity of endrin to mammals is greater than that of other organochlorine pesticides (Sect. 6.3.3). Sublethal effects in wild animals manifest primarily as disorders of behavior and reproduction; improper maternal care, temporary loss of normal activity, and increased vulnerability to predators are examples of the former, while reduced reproductive potential, increased postnatal mortality, and fetal death typify the latter (Sect. 5.5.2).

Laboratory studies have revealed an extensive array of adverse effects in mammals following chronic exposure to endrin (Sect. 6.3.3). Endrin can affect the central nervous system at doses substantially below lethal or convulsive levels. For example, electroencephalogram changes were observed in squirrel monkeys which received a total endrin dose of 1 mg/kg; seizures were not observed until 10 mg/kg. Exposure of a variety of species to various doses of endrin via several routes produced diffuse degenerative changes in one or more of the following organs: liver, kidney, heart, brain, lung, adrenal glands, and spleen. Inhalation of 0.36 ppm for 118 seven-hour periods was sufficient to produce pneumonitis in rabbits. Ingestion of 3 ppm for 19 months resulted in enlargement of the kidneys and heart in dogs, while 5 ppm for two years produced increases in liver weight in rats (Sect. 6.3.3). Additional effects included blindness, hypertension, bradycardia, changes in carbohydrate metabolism, abnormalities of electron transport, abnormal trace-metal mobilization, changes in blood composition, lethargy, avoidance of food, weight loss, vomiting, and others (Sect. 6.3.3).

No malignancies attributable to endrin exposure have been reported in the literature; however, endrin has been found to cause chromosomal aberrations in rats following intratesticular injection (Sect. 6.3.4). Teratogenesis, growth retardation, and increases in fetal and postnatal mortality have been observed in rats and hamsters following ingestion of endrin (Sects. 6.3.4 and 6.3.5).

Chronic exposure to endrin may also be fatal. Five to eight ppm in the diet was fatal to dogs in 18 to 44 days. Twelve ppm in the diet for life decreased the survival time for rats. Deer mice succumbed to a diet which contained only 2 ppm endrin (Sect. 6.3.3).

Development of endrin resistance has been observed in pine mice (*Pitymys pinetorium*). Resistance was shown to be hereditary as well as acquired (Sect. 5.5.2).

Human exposure to endrin occurs through the diet, through inhalation, and through dermal contact. The average dietary intake in the United States in 1973 was 0.033 μg/day for a 69.1-kg man. This level is far below the maximum acceptable daily intake of 138.2 μg/day established by the World Health Organization (Sect. 8.3.1). Respiratory and dermal exposure to endrin is possible during manufacture and distribution, but is more likely to result, directly or indirectly, from agricultural uses (Sect. 6.2.1).

Humans do not tend to store endrin in significant quantities, and residues are only detected in human tissue immediately after an acute exposure. Humans fatally poisoned by endrin have been reported to have an endrin content as high as 400 ppm in fat and 10 ppm in other tissues (Sect. 6.2.2).

Outbreaks of human poisoning have resulted from accidental contamination of foods and have been traced to doses as low as 0.2 mg/kg body weight. The toxicity of endrin appears to result primarily from the effects of endrin on the central nervous system. Symptoms usually observed in victims of endrin poisoning were convulsions, vomiting, abdominal pain, nausea, dizziness, and headache. When death occurred, respiratory failure was the most common cause. Sixty fatalities resulted following ingestion of 5 to 50 mg/kg during a poisoning episode in India.

Men employed in the manufacture of endrin show significantly increased activity of hepatic microsomal enzymes. No other adverse effects of occupational exposure to endrin have been reported (Sect. 6.3.1).

Due to the interdependency of all compartments of the ecosphere, the adverse effects of endrin on fish and wildlife inevitably spread to man. The most direct consequence of release of endrin into the environment is contamination of the human food supply. Humans ingest endrin-treated agricultural produce as well as meat from steers, lambs, and hogs that feed on contaminated vegetation. Ingestion of 20 mg endrin per day by cows results in detectable quantities of endrin in milk; levels of 0.25 ppm endrin in milk on a fat basis have been observed. Aquatic invertebrates and fish may bioconcentrate considerable quantities of endrin from water and pass the endrin onto predator birds. The contaminated fowl (or the fish themselves) may, in turn, be ingested by humans (Sect. 8.3).

Although contamination of food by endrin still occurs to some extent, it is apparently decreasing. The frequency of endrin occurrence in food in 1973 (0.3%) was considerably less than the average frequency of occurrence over the six-year period 1965 to 1970 (2.05%). At present, levels are 4000 times lower than those acceptable to the World Health Organization. Endrin therefore does not pose a hazard to the general population, and concern over the possible occurrence of levels capable of threatening human health is needed only in the geographical locations where the pesticide is used extensively.

1.2 MAJOR CONCLUSIONS

1. The use of endrin is confined, primarily, to the southeastern and delta states of the United States.

2. Environmental contamination by endrin appears, for the most part, to be restricted to those areas where endrin is used intensively.

3. Background concentrations in the atmosphere, hydrosphere, and lithosphere, far removed from agricultural areas where endrin is applied and industrial areas where endrin is manufactured, are generally below the levels of detection.

4. Transport of endrin to remote areas and to the open ocean is expected to occur primarily via the atmosphere.

5. Monitoring data for endrin in the atmosphere are limited due to the difficulties inherent in ambient air sampling.

6. Endrin is dissipated from the environment by photochemical and thermal decomposition and by microbial degradation.

7. The major product of endrin decomposition under most natural conditions is ketoendrin.

8. Relatively few bacteria are capable of degrading endrin, and degradation occurs most readily under anaerobic conditions.

9. Many species of algae and fungi are capable of endrin degradation.

10. Bioaccumulation of endrin has been observed in blue-green algae, and growth inhibition has been observed in various species of algae and phytoplankton following exposure to endrin.

11. Vascular plants absorb, translocate, and metabolize endrin.

12. Endrin generally is nontoxic to plants.

13. Aquatic invertebrates and fish bioconcentrate significant quantities of endrin from the water.

14. Endrin is more toxic to aquatic arthropods than to mollusks and is the most toxic to fish of all the commonly used organochlorine insecticides.

15. The primary effects of endrin on fish are disorders of the central nervous system, culminating in death.

16. Avians are exposed to endrin primarily through ingestion of contaminated aquatic organisms and water.

17. Endrin is extremely toxic to birds, and lethality is the most frequently observed effect.

18. The basis of endrin toxicity in insects has not yet been determined, but the central nervous system is believed to be the site of action.

19. Endrin is highly toxic to all mammals regardless of the route of exposure.

20. Endrin does not persist in mammals, since it is rapidly degraded and eliminated.

21. Acute exposure of mammals results in central nervous system disorders, with convulsions leading to death through respiratory failure within two days.

22. Chronic exposure may result in damage to the liver, kidneys, heart, brain, lung, adrenal glands, and spleen.

23. Behavioral abnormalities, reproductive disorders, changes in carbohydrate metabolism, changes in blood composition, and other effects secondary to central nervous system disorders have been observed following chronic exposure of mammals to sublethal doses of endrin.

24. While no malignancies attributable to endrin have been reported, chromosomal abnormalities and teratogenesis have been induced by endrin in several mammalian species.

25. Endrin residues have only been detected in human tissue immediately after an acute exposure.

26. Symptoms of acute endrin exposure in humans include convulsions, vomiting, abdominal pain, nausea, dizziness, and headache, with death resulting from respiratory failure.

27. The effects of chronic endrin exposure in humans remain speculative.

28. Human exposure to endrin from ingestion of contaminated food is decreasing.

29. The possibility of the food supply being contaminated to an extent that it poses a threat to human health exists only in those areas where endrin is used extensively.

2. PHYSICAL AND CHEMICAL PROPERTIES AND ANALYSIS

2.1 SUMMARY

Endrin, a member of the cyclodiene insecticides, is a cyclic hydrocarbon having a chlorine-substituted methanobridge structure. It is synthesized by the oxidation of isodrin at temperatures below 100°C. The product is then normally stabilized by the addition of hexamethylene tetramine to remove traces of acid, which are responsible for spontaneous exothermic conversions.

Technical-grade endrin is a light tan powder, which is stable in the presence of oxidizing agents and alkaline reagents. However, exposure to acids or sufficient heat can catalyze molecular rearrangement.

Since the only methods for removal of endrin from the environment appear to be microbial degradation, photodecomposition, and thermal decomposition, the last two physical liabilities of endrin are quite significant to environmental distribution and longevity.

Many techniques for endrin determination have been reported, including bioassay, total chlorine content, silylation, acetylation, spectrophotometry, and colorimetry. The most sensitive qualitative and quantitative methods available involve variations in gas chromatography.

2.2 NOMENCLATURE, STRUCTURE, AND SYNTHESIS

Endrin (1,2,3,4,10,10-hexachloro-6,7-epoxy-1,4,4a,5,6,7,8,8a-octahydro-1,4-endo, endo-5,8-dimethanonapthalene) is the common name for one member of the cyclodiene group of insecticides (Shell Chemical Company, 1959). The cyclodiene insecticides are cyclic hydrocarbons having a chlorine-substituted methanobridge structure.

Endrin contains at least 92% of the endo-endo stereoisomer of dieldrin or the epoxide of isodrin. Compound 269 and Mendrin are two additional names given to endrin; the former was used by the Hyman Company during its developmental stages, and the latter is the trade name used in South America and India (Shell Chemical Corporation Handbook, 1959).

The two most common technical systems used in naming endrin are the British and American. In the British system the prefixes exo and endo are applied to the methanobridges according to positioning outside or inside the bent cage structure as a whole. The first prefix that appears in the name refers to the methanobridge of the lowest numbered nucleus (1 through 4), while the second prefix applied to the methanobridge is the highest numbered nucleus (5 through 8). Thus, following the British system of nomenclature, the chemical name of endrin is 1,2,3,4,10,10-hexachloro-6,7-epoxy-1,4,4a,5,6,7,8,8a-octahydro-exo-1-4,exo-5,8-dimethanonaphthalene.

In the American system the terms exo and endo indicate which pairs of exo or endo bonds of each of the norbornene nuclei are used to fuse the two ring systems together; each prefix, therefore, defines the stereochemistry of the norbornene nucleus to which it applies (Soloway, 1955). Accordingly, the chemical name for endrin is 1,2,3,4,10,10-hexachloro-6,7-epoxy-1,4,4a,5,6,7,8,8a-octahydro-1,4-endo,endo-5,8-dimethanonaphthalene.

Both of these systems of nomenclature name endrin as a derivative of octahydro-dimethanonaphthalene. The structural differences between endrin and other members of the cyclopentadienes are indicated by the use of the terms exo and endo (relative position of methanobridges in each compound). Benson (1969), recognizing that none of the compounds of the cyclopentadiene series was aromatic, recommended the use of the von Baeyer system, which is widely used and endorsed by the International Union of Pure and Applied Chemistry (IUPAC) in naming the polycyclic aliphatic compounds. According to the von Baeyer-IUPAC system, endrin is named as a derivative of 2,3,7,6-endo-2,1-7,8-endo tetracyclo $(6.2.1^{3,6}.0^{2,7})$ dodeca-9-ene, which reflects the fact that it originally received its name because the two fused norbornene ring systems present were endo-endo with respect to one another. Therefore, Bedford (1974), using the von Baeyer-IUPAC system for numbering and naming polycyclic aliphatic compounds, named endrin as the 1,8,9,10,11,11-hexachloro-4,5-exoepoxy-2,3,7,6,-endo-2,1,7,8-endo tetracyclo $(6.2.1^{3,6}.0^{2,7})$ dodec-9-ene, which corresponds to the structure shown in Fig. 2.1.

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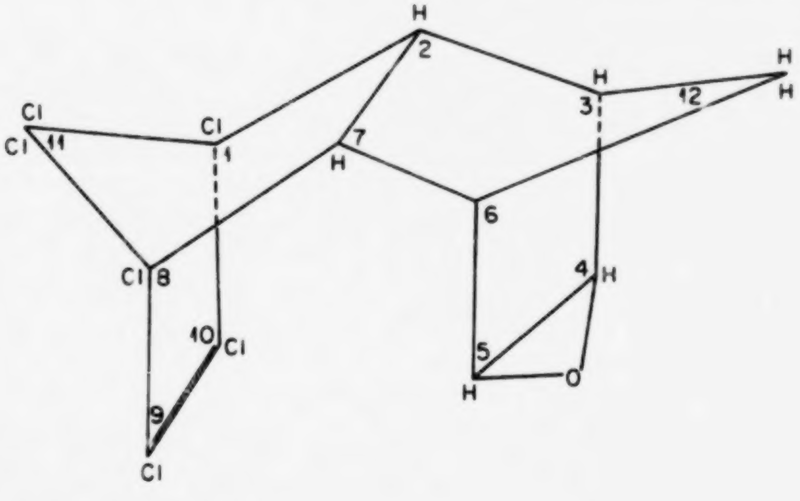

Fig. 2.1. Stereochemical structure of endrin, using the von Baeyer-IUPAC notation (1,8,9,10,11,11-hexachloro-4,5-exoepoxy-2,3,7,6-endo-2,1,7,8-endo tetracyclo $[6.2.1^{3,6}.0^{2,7}]$ dodec-9-ene).

For the purpose of this review, the American system of nomenclature will be used.

The chlorinated component (left side) of the endrin molecule, which represents 75% of its mass, is structurally identical to the other members of the cyclodiene group, including alodan, aldrin, dieldrin, isodrin, heptachlor, chlordane, and isobenzan.

Roll et al. (1969) applied the nuclear quadrupole resonance (NQR) technique in order to determine the structure of endrin. The NQR spectrum of endrin was found to exhibit five major resonance frequencies. The resonance of both vinylic chlorine atoms appeared at 37.7 MHz, the bridge dichloromethylene chlorine atom resonances occurred at 36.3 and 37.0 MHz, and the remaining bridgehead chlorine atom resonances were found at 35.9 and 36.1 MHz.

The endo-endo characteristics of endrin were substantiated by the work of Keith (1971), who, in an effort to study the degradation reactions of some pesticides, implemented the use of paramagnetic resonance spectroscopy (PMR), which employs a chemical-shift reagent $Eu(DPM)_3$, tris(dipivalomethanato europium). Previously, Keith and co-workers (1970) found that the special feature of the PMR spectra of endrin was the splitting of the H_2, H_7 signal into a pair of doublets due to the coupling of H_2 with H_3 and H_6 and the coupling of H_7 with H_6 and H_3. However, this characteristic of the endo-endo ring fusion was obscured by the overlap of the $H_{2,7}$ and the $H_{4,5}$ signals of endrin in CCl_4. The addition of $Eu(DPM)_3$ to the endrin solution shifted both the $H_{2,7}$ and $H_{4,5}$ signals down field in such a way that the $H_{4,5}$ shift was much greater in magnitude than that of the $H_{2,7}$. By separating these two signals, the splitting of the H_2, H_7 signal at the 250 H_2 sweep width is easily observed.

The application of ^{13}C nuclear magnetic resonance (natural abundance, Fourier transform spectroscopy) as a means of establishing the complete molecular configuration of endrin (including the chlorinated section of the molecule) was undertaken by Roberts et al. (1974). The confirmation of the resonance positions of the different carbon atoms in the NMR spectra was successfully attained by the use of the chemical-shift reagent tris(1,1,1,2,2,3,3-heptafluoro-7,7-dimethyl-4,6-octanedionate) europium(III), $Eu(FOD)_3$, as suggested by Rondeau and Sievens (1971).

Endrin is produced by the oxidation of isodrin, with hydrogen peroxide in acetic acid, at the lowest temperature possible (Fig. 2.2). At 100°C, isodrin rearranges to a ketone that has no insecticidal effect (Melnikow, 1971).

The precursor of endrin, isodrin, is prepared by the condensation of cyclopentadiene with 1,2,3,4,7,7-hexachlorobicyclo-(2.2.1)-2,5-heptadiene according to the reaction shown in Fig. 2.3, whereas the 1,2,3,4,7,7-hexachlorobicyclo-(2.2.1)-2,5-heptadiene is produced by reacting hexachloropentadiene with vinyl chloride, followed by dehydrochlorination with an inorganic base (Fig. 2.4).

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ISODRIN (1) + ACETIC ACID-HYDROGEN PEROXIDE ⟶ ENDRIN

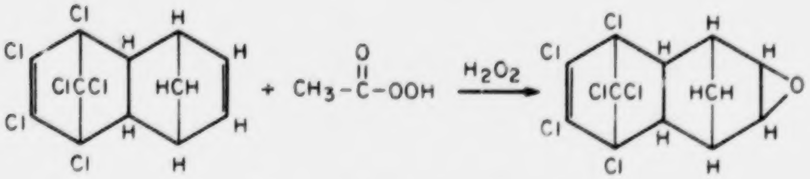
Cl
Cl
Cl
Cl
ClCCl
H
H
H
H
H
H
HCH
O
CH3-C-OOH
H2O2
Cl
Cl
Cl
Cl
ClCCl
HCH
H
H
H
H
H
H
O

Fig. 2.2. Synthesis of endrin.

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1, 2, 3, 4, 7, 7 — HEXACHLOROBICYCLO-(2.2.1)-2,5-HEPTADIENE +
CYCLOPENTADIENE ⟶ ISODRIN

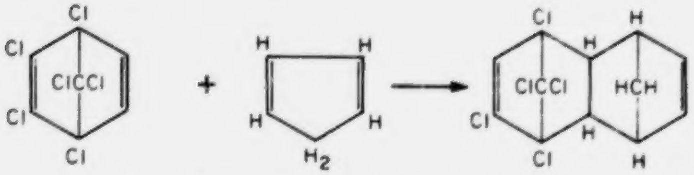
Cl
Cl
Cl
Cl
ClCCl
H
H
H
H
H2
Cl
Cl
Cl
ClCCl
HCH
H
H
H
H

Fig. 2.3. Synthesis of isodrin.

ORNL-DWG 79-8141

1, 2, 3, 4, 5, 7, 7 - HEPTACHLOROBICYCLO-(2.2.1)-HEPT-2-ENE +
INORGANIC BASE ⟶ 1, 2, 3, 4, 7, 7-HEXACHLOROBICYCLO-(2.2.1)-
2, 5 — HEPTADIENE

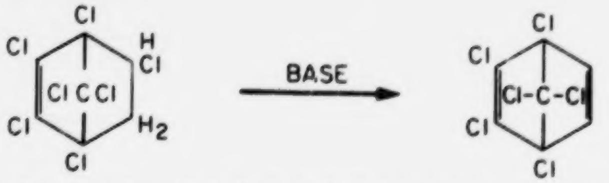
Cl
Cl
Cl
Cl
H
Cl
Cl C Cl
H2
BASE
Cl
Cl
Cl
Cl
Cl-C-Cl

Fig. 2.4. Synthesis of 1,2,3,4,7,7-hexachlorobicyclo-(2.2.1)-2,5-heptadiene.

The endo-endo isomers, like endrin, are less stable than the endo-exo isomers such as dieldrin. Therefore, endrin formulations are stabilized by the addition of small amounts of hexamethylene tetraamine (Shell Chemical Corporation, 1959), inorganic or organic nitrites, epichlorohydrin and glycidyl ethers (Bellin, 1956), or various alkaline-metal carbonates (urea, etc.) to remove traces of acid that are responsible for spontaneous exothermic conversions.

The composition of a typical production batch of endrin as reported by Shell Chemical Corporation (1959) is shown in Table 2.1.

Table 2.1. Chemical composition of technical endrin

Compound	Percent (wt)
Endrin	96.6
Dieldrin	0.42
Aldrin	0.03
Isodrin	0.73
1,2,3,4,7,7-Hexachloro-1,4-dihydro-1,4-methanobenzine	0.03
1,2,3,4,5,7,7-Heptachloro-1,4,5,6-tetrahydro-1,4-methanobenzene (heptachloronorbornadiene)	0.08
1,2,3,4,5-Pentachloro-7-oxo-1,4,5,6-tetrahydro-1,4-methanobenzene (heptachloronorborn[illegible])	0.09
Endrin half-cage ketone (Δ-ketoendrin; $C_{12}H_8Cl_6O$)	1.57
Endrin aldehyde ($C_{12}H_8Cl_6O$)	<0.05
Acidity (calculated as HCl)	0.18
Unidentified components	0.12

Source: Shell Chemical Corporation, 1959.

2.3 PHYSICAL AND CHEMICAL PROPERTIES

Chemically pure endrin is a white crystalline solid, while technical-grade endrin is a light tan powder (Brooks, 1974a). Endrin is described as being insoluble in water, only 0.19 ppm (Robeck et al., 1965), sparingly soluble in methanol, of relatively low solubility in aliphatic hydrocarbons (0.3 lb/gal), and quite soluble in aromatic solvents (3.0 lb/gal) (Brooks, 1974a).

Technical-quality endrin, as well as the purified cyclodiene, is stable in the presence of wetting agents, emulsifiers, alkaline oxidizing agents, and basic reagents. However, the endo-endoconfiguration is vulnerable to molecular rearrangement catalyzed by acids or heat. Endrin will melt with this consequent decomposition at 200°C. Therefore, endrin is susceptible to some carriers used in synthesis, and this decomposition mechanism is deactivated by the addition of hexamethylenetetramine.

Chemically pure endrin is stable upon exposure to either light or air. However, exposure to Lewis acids, heat, or ultraviolet radiation induces the formation of aldehydes, alcohols, or ketones.

Endrin is nonflammable (Robeck et al., 1965), and it has a vapor pressure of 2×10^{-7} mm Hg at 25°C. A specific gravity of 1.7 at 20°C is also typical of the compound (Brooks, 1974a).

The chlorine content of endrin accounts for between 55 and 57% of its molecular weight, which is 380.9. Endrin contains less than 0.1 wt % water and less than 0.4 wt % free acid (calculated as acetic acid). Less than 0.5% of endrin remains as an insoluble residue after solubilizing in xylene (Brooks, 1974a).

Table 2.2 contains a compilation of the physical and chemical properties of endrin.

Table 2.2. Physical and chemical properties of endrin

Color	White (purified grade) Light tan (technical grade)
Physical state	Crystalline solid (purified grade) Powder (technical grade)
Melting point	200°C (decomposition)
Vapor pressure	2.7×10^{-7} mm Hg at 25°C
Specific gravity	1.7 at 20°C
Chlorine content	55 to 57 wt %
Water content	0.1 wt %
Free acid content	0.4 wt % (acetic acid)
Solubility	31 g/100 ml acetone at 25°C 28 g/100 ml amylacetate 51 g/100 ml benzene 51 g/100 ml carbon tetrachloride 10 g/100 ml diesel oil 3 g/100 ml ethanol 87 g/100 ml ethylene dichloride 10 g/100 ml fuel oil 3 g/100 ml isopropanol 5 g/100 ml kerosene 2 g/100 ml methanol 40 g/100 ml methyl ethyl ketone 74 g/100 ml toluene 21 g/100 ml turpentine 55 g/100 ml xylene

Source: Reprinted with permission from G. T. Brooks, *Chlorinated Insecticides*, Vol. 1, Technology and Application, 1974. Copyright The Chemical Rubber Co., CRC Press, Inc.

Endrin reacts to give products of simple ring opening with both neutral and acidic agents (Lidov et al., 1950). Soloway et al. (1960) obtained a significant product of endrin rearrangement by refluxing for 23 hr (with vigorous stirring) a mixture of 25 g endrin, 100 ml butyl ether, and 100 g concentrated hydrochloric acid. The new compound, a pentacyclic ketone (Fig. 2.5, structure 10), was also obtained by heating 2 g endrin in a test tube at 150°C. The exothermic reaction occurred when the temperature rose to 178°C. In the acid-catalyzed rearrangement, the conjugate acid of endrin was said to undergo ring opening of the oxirane group with the formation of a bond between carbons 4 and 10. The extra charge left on carbon 9 is stabilized by a hydride shift from carbon 5 and a loss of a proton from oxygen to give the pentacyclic ketone.

Skerrett and Baker (1959) treated endrin with boron trifluoride in benzene solutions and obtained two separate compounds, the half-cage ketone (Fig. 2.5, structure 3) and the pentacyclic aldehyde (Fig. 2.5, structure 2). On the other hand, Weincke and Burke (1969) showed that treatment of endrin with zinc chloride in hydrochloric acid converted endrin to the half-cage ketone without any other by-products (Fig. 2.5, structure 3).

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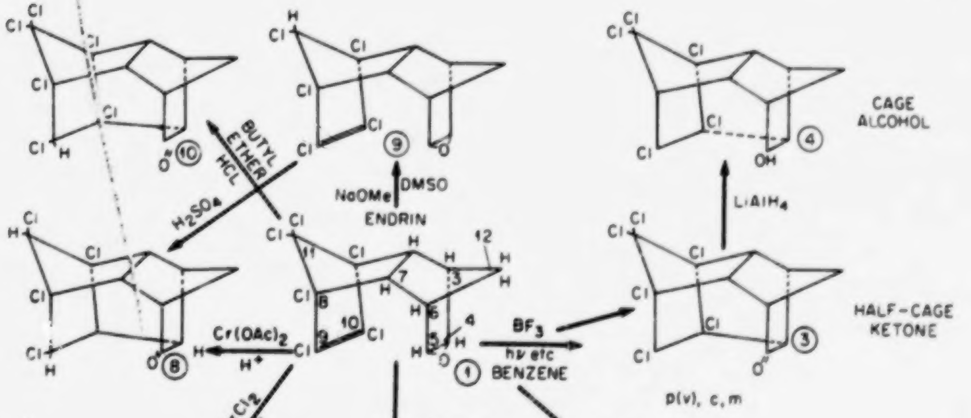


BUTYL ETHER HCL
H2SO4
NaOMe
DMSO
ENDRIN
Cr(OAc)2
H+
CrCl2
H+
hν HEXANE
BASE
BF3
hν etc
BENZENE
LiAlH4
CAGE ALCOHOL
HALF-CAGE KETONE
PENTACYCLIC ALDEHYDE
p(v), c, m
m e
CH=O
OH
Cl
H
①
②
③
④
⑤
⑥
⑦
⑧
⑨
⑩

Fig. 2.5. Schematics of some chemical reactions of endrin.

Further treatment of the ketone with lithium aluminum hydride produced the cage alcohol as shown in Fig. 2.5, structure 4. Brooks (1974a), utilizing the results of Adams and Mackenzie (1969), suggested that endrin was reductively dechlorinated with sodium methoxide in methanol-dimethylsulfoxide in order to give analogues in which one of the chlorine atoms in the methanobridge was replaced by hydrogen (Fig. 2.5, structure 9); presumably, this conversion occurred without skeletal rearrangement.

In connection with derivative formation for identification of endrin residues by gas chromatography, Chau and Cochrane (1969) found that treatment with concentrated sulfuric acid at room temperature for 10 to 15 min caused the isomerization of endrin to an aldehyde and a ketone (Fig. 2.5, structures 2 and 3). Further investigations into derivative formation (Chau and Cochrane, 1971) produced the reduction of endrin by chromous chloride to yield a pentachloro derivative whose elemental analysis, IR and NMR spectroscopy, suggested structure 7, Fig. 2.5, but treatment of endrin with chromous acetate under acidic conditions produced small amounts of the isomeric ketone (Fig. 2.5, structure 8) (Chau, 1972). Endrin also reacts with bromine to form the 4,5-dibromide, which, in turn, reacts with sodium sulfide enneahydrate to form the 4,5-episulfido compound, producing the sulfoxide upon oxidation (Metcalf, 1955).

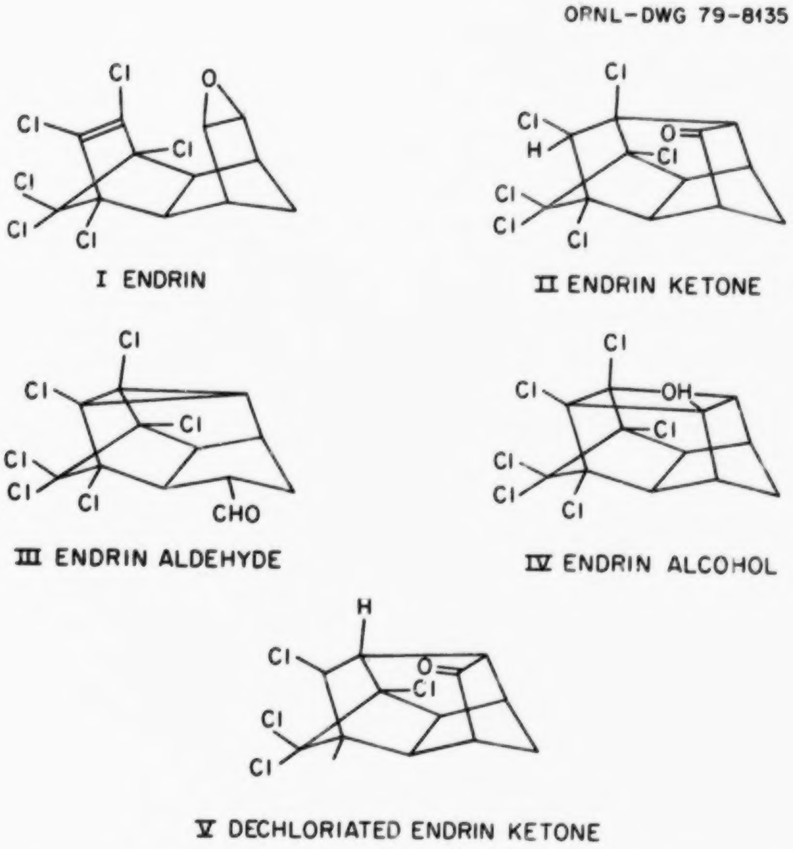
The electrochemical reduction of endrin was studied by Bukowsky and Cisak (1968). Their results indicated that the 11-endo chlorine was first to be replaced followed by both chlorines in positions 9 and 10. The bridgehead chlorines (positions 1 and 8), which are equivalent, were the last to be replaced.

2.4 IMPLICATIONS OF CHEMICAL AND PHYSICAL PROPERTIES FOR BEHAVIOR OF ENDRIN IN ORGANISMS, MEDIA, STORAGE, AND AGRICULTURE

In order to determine the chemical nature of toxicity, Soloway (1965) examined the insecticidal action of 106 cyclodienes. High insecticidal activity was found only in those compounds with two centers of electronegativity placed close to each other and situated in the plane of symmetry as defined by the dimethanobridge. Consequently, there were very small differences in insecticidal action among aldrin, dieldrin, endrin, and telodrin. The similarities in biological effects caused by these pesticides on the central nervous systems suggest that all of them act on the same sites. According to Benson (1969), the electron-rich sites of these molecules, which were not believed to be chemically active, are most likely locations for strong electrostatic interaction possible on nerve cell membranes.

Further results of Soloway (1965) suggested that the epoxides were the active compounds, while only those other compounds in which the double bond of the nonchlorinated side of the molecule is readily epoxidized display insecticidal activity. The rigid-cage configuration of the cyclodiene compounds possessing electronegative centers in their chlorine atoms and their lipid-soluble characteristics is believed to be important to their passage across membranes as well as their molecular action (Hathway, 1965).

The major products resulting from the photochemical and thermochemical isomerization of endrin have been identified, and their structures, along with that of the parent compound, are presented in Fig. 2.6. Since these two processes and microbial decomposition are the only means of endrin degradation, they are significant parameters for environmental distribution.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I ENDRIN

II ENDRIN KETONE

III ENDRIN ALDEHYDE

IV ENDRIN ALCOHOL

V DECHLORIATED ENDRIN KETONE

Fig. 2.6. Some thermal and photochemical degradation products of endrin. Source: modified from Plimmer, 1971.

Photolysis of solid endrin (I) by exposure to a germicidal lamp gave a 37% yield of endrin ketone (II) and a 9% yield of the aldehyde (III). These compounds together with a small quantity of the "bird-cage" alcohol (IV) have also been identified following the thermal isomerization of endrin (Plimmer, 1971). Photolysis of endrin in hexane gives a monodechlorinated ketonic compound (V). The dechlorinated endrin isomer has been identified in the field as well (Klein and Korte, 1967).

Mitchell (1962) and Roburn (1963) accomplished the photochemical isomerization of endrin by exposing samples of the pesticide to ultraviolet light. Rosen et al. (1966), extending Roburn's work, found that exposure of endrin to a 2537-A germicidal lamp for 48 hr yielded 37% ketone and 9% aldehyde. The infrared spectra of these two isomers were identical to those published by Phillips et al. (1962). The mechanism of isomerization of these two carbonyl compounds from endrin involves either a hydride shift or hydrogen abstraction of an epoxy hydrogen, while photosensitizers such as rotenone appear to enhance the photochemical alteration (Ivie and Casida, 1970). Zabik et al. (1971), using a somewhat different approach, studied the photolytic decomposition of endrin in solution. Hexane and cyclohexane solutions of endrin were exposed to uv sources (253.7 to 300 mm) and sunlight. The extent of the reactions were followed by both gas chromatography and IR spectroscopy. Irradiation of hexane solutions of endrin gave essentially the same results as those obtained with cyclohexane. The photolysis of endrin, using uv light or sunlight, proceeded with the formation of a half-cage ketone compound 1,8-exo-9,11,11-pentachloro-pentacyclo-$(6.2.1.1.^{3,6}.0^{2,7}.0^{4,10})$ dodec-5-one. This photoproduct of endrin was found to be highly resistant to the usual oxidation and reduction processes (Fig. 2.5, structure 5). However, the only significant reaction of the molecule was that of a trans-annular enolization (homoenolization) in the presence of bases to give the pentachloro-cage alcohol. Its suggested structure based on IR, uv, and NMR spectroscopy is shown in Fig. 2.5, structure 6.

The rate of photoconversion of endrin in the solid state to its isomerides on exposure to natural sunlight was studied by Burton and Pollard (1974). Individual glass planchets containing 10 mg endrin were exposed to natural sunlight. At intervals, samples were removed from the sunlight and analyzed for the disappearance of endrin and for the appearance of ketoendrin by infrared spectroscopy. The experiment was conducted once in June and once in October in order to assess the effects of sunlight intensity of the reaction rate. The results for both trials are presented in Table 2.3 and in Figs. 2.7 and 2.8. The intensity of the sunlight is apparently a very important factor in determining the rate of isomerization, since the disappearance of endrin and the conversion to ketoendrin were considerably more rapid in June than in October. The change in the slope of the curves in Fig. 2.7 after nine days is also indicative of a decreasing rate of isomerization and probably reflects the diminishing intensity of the October sunlight toward the end of the month.

Table 2.3. Endrin photoproduct formed vs sun's intensity

Days exposed	Month	Isomeric ketone (II) formed (%)
5	October	14
5	June	46
12	October	30
12	June	65

Source: W. B. Burton and G. E. Pollard, *Bull. Environ. Contam. Toxicol.* 13(1): 113-116 (1974). Copyright 1974 by Springer-Verlag.

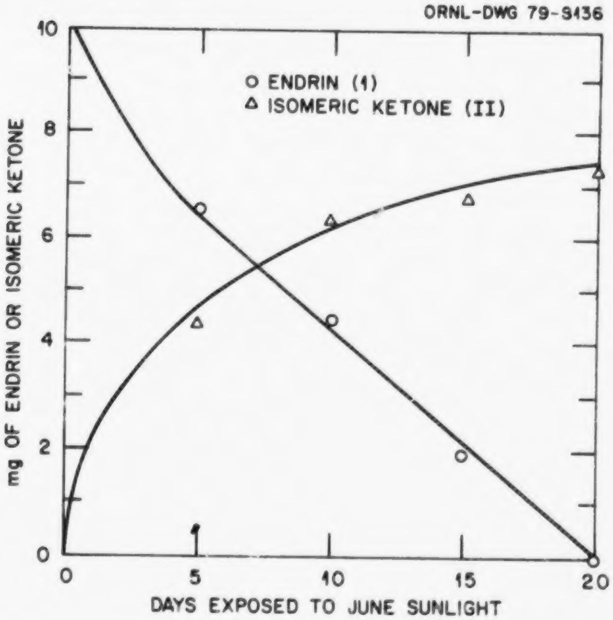

Fig. 2.7. Photochemical isomerization of endrin. Source: W. B. Burton and G. E. Pollard, *Bull. Environ. Contam. Toxicol.* 13(1): 113-116 (1974). Copyright 1974 by Springer-Verlag.

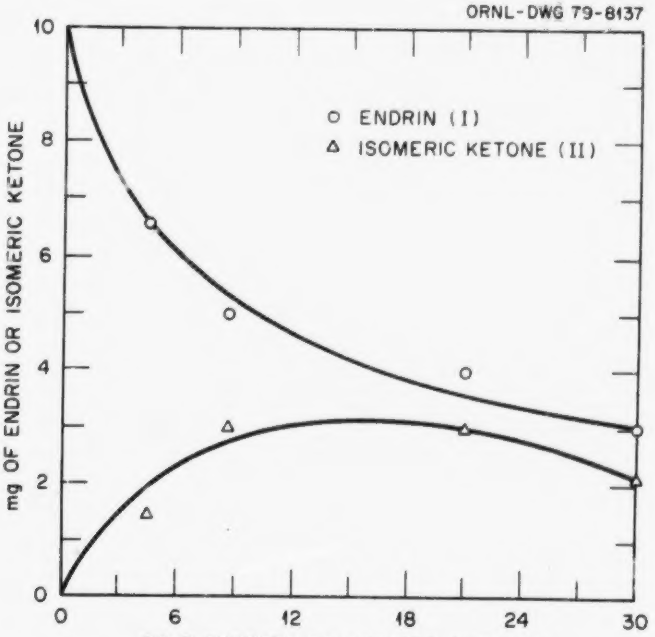

Fig. 2.8. Photochemical isomerization of endrin. Source: W. B. Burton and G. E. Pollard, *Bull. Environ. Contam. Toxicol.* 13(1): 113-116 (1974). Copyright 1974 by Springer-Verlag.

Phillips et al. (1962), in studying the gas chromatographic behavior of endrin at 230°C, found that this compound gave two peaks of almost equal size and that neither one of the two peaks was due to endrin. In order to evaluate this finding, endrin was placed in a potassium bromide dish and heated to 240°C. Infrared spectra, as well as gas chromatographic analysis of the reaction products, showed that the two peaks found in the gas chromatographic analysis of endrin corresponded to two different compounds, namely, a ketone and an aldehyde (Fig. 2.5, structures 2 and 3).

The isomerization of endrin to ketoendrin over a five-year dark storage period at room temperature was reported by Barlow (1965). Apparently, the isomerization process at room temperature was triggered by impurities present in the endrin sample. Later studies carried out by Graham and Kenner (1969) showed that heat-induced decomposition of endrin in KBr dishes or in the open followed the same route, regardless of temperature, to give a large excess of the ketone isomer over the aldehyde isomer. This finding led to the recommendation that endrin standards should be stored in opaque bottles below room temperature, and that standards should be evaluated for composition before use.

2.5 METHODS OF ANALYSIS

The preparation of samples prior to endrin extraction varies according to the type of material to be analyzed. In general, pulverized or dust formulations require minimal preparation, whereas human, animal, or plant tissues are minced or blended into homogenates. Appropriate sample aliquots are then extracted with solvents such as acetone, benzene, toluene, hexane, petroleum, ether, pentane, or any combination of these polar-nonpolar solvents (Chau, 1972b; Reinke et al., 1973; Bontoyan and Jung, 1972; Stalling et al., 1972; Young and Burke, 1972; Ali et al., 1969; Goodwin et al., 1961).

Endrin extraction is accomplished by either maceration of the samples, exhaustive extraction in a Soxhlet apparatus, or dynamic equilibration (shaking) for a predetermined period of time. Elution of the sample through a chromatographic column is also used and recommended in some analytical procedures; however, the extraction procedure used is dependent upon the type of material to be analyzed. Fibrous materials such as plant tissues require longer contact with the extracting solvent than do powders or dust formulations.

The treatment of the extract for qualitative or quantitative measurement of endrin is determined by the nature of the extract, the method of detection, and the desired accuracy. In a few cases it is possible to use an analytical method in which there is little or no interference from impurities (Biros et al., 1972; Dougherty et al., 1972). In most cases, however, some degree of purification is necessary, and the cleanup procedures include distillation, evaporation, solvent fractionation, and partition chromatography through florisil, celite, silica gel, or aluminum oxide columns.

Partition of pesticide extracts between immiscible solvents is often used to give partition cleanup before florisil chromatography. Seven of these liquid-liquid partition techniques are fully described in the Food and Drug Administration pesticide manual (1971).

A partitioning method for sample cleanup prior to gas-liquid chromatography (GLC) determination of common organochlorine pesticide residues in biological samples was described by Kadoum (1969). Plant or animal tissue hexane extracts are partitioned four consecutive times with 95% aqueous acetonitrile solutions. The total recovery of endrin, measured by peak height in the chromatogram, amounted to 95% of that added to the biological extract. Young and Burke (1972) used a microscale alkali treatment on biological sample extract prior to endrin determination by GLC. Endrin extracts were refluxed with alcoholic potassium hydroxide in the steam bath for a predetermined length of time and partitioned into hexane. Recoveries of unchanged aldrin, dieldrin, and endrin range from 70 to 90%. Young suggested that the high recovery following alkali treatment be used in the identity confirmation of these pesticides.

The troublesome lipid removal from fish extracts prior to GLC was successfully accomplished by gel permeation chromatography. Using cyclohexane and Bio Beans SX2, Stalling et al. (1972) obtained 100% recoveries of ^{14}C-endrin with less than 0.5% of the original lipid present in the endrin fraction. In the case of fish extracts containing endrin at concentrations below 0.1 ppm, additional cleanup with florisil was recommended.

The analytical methods used for quantitative or qualitative determination of endrin fall into two categories. The first consists in using the ability of living organisms to withstand the toxicological effects of powders, dust formulations, emulsifiable liquids, and extracts of endrin. These techniques are classified as biological methods or bioassays. The second category utilizes one or more physicochemical properties of endrin as well as any available analytical instrumentation designed for the measurement of those properties. These methods are classed as chemical methods.

Biological methods using fish, insect larvae, aphids, and daphnia have been described in the literature by Ali et al. (1969, 1973), Tew and Sillibourne (1961a, 1961b), McDonald (1962), and Sun and Sanjean (1961). All of these methods use either percent mortality or LD_{50} data obtained by submitting the test organisms to unknown concentrations of purified or nonpurified endrin extracts. The results obtained are quantified by comparison with mortality or LD_{50} data produced by known concentrations of endrin under the same experimental conditions. Although these methods are convenient and relatively inexpensive, they lack sensitivity, specificity, and, in some cases, reproducibility.

The determination of total chlorine is one of the most common chemical methods for analysis of chlorinated pesticides. An application of this technique to endrin analysis is described by Porter (1964). There are many modifications to this technique, but the usual Stephanov's procedure involves three stages: (1) refluxing the sample for half an hour with metallic sodium in 99% isopropyl alcohol, (2) elimination of excess sodium, and (3) titration of chlorine by the classical Volhart method.

As an alternative to the sodium reduction procedure, the Association of Official Analytical Chemists (AOAC) methods book (1975) uses sodium diphenyl to liberate chlorine ions from endrin samples. In a still different analytical procedure, endrin is treated with H_2SO_4 to form a ketone derivative which possesses a definite and identifiable retention time in the GLC (Chau and Cochrane, 1969). Similarly, HBr, CrO_3, and HCl under various conditions have been utilized for the positive identification of endrin found in soil, vegetables, forage crops, and fat extracts (Chau and Cochrane, 1971). An alternative confirmation test for endrin utilizes preferential reductive dechlorination of endrin with $CrCl_2$ in order to yield the pentachloroketone. This derivative has a different retention time from the common organochlorine pesticides (Chau, 1970).

Woodham et al. (1972) found that endrin will form derivatives when reacted with 10% BCl_3-2-chloroethanol solution. The new derivative delta-ketoendrin produced a characteristic GLC peak with longer retention time, while the reaction time required for this conversion was only 10 min. On the other hand, Chau's (1974) simplified procedure for the acid-catalyzed isomerization of endrin uses acidified alumina microcolumns as the solid matrix for the reaction to occur. Yields of 75% ketone I isomer were obtained in approximately 1-1/2 hr contact time, and no interference from chlorinated pesticide residues other than dieldrin was found to occur. This procedure is sensitive to 1 mg endrin.

Silylation and acetylation reactions for the identification and confirmation of the photoproducts of endrin by GLC were defined by Chau (1972b). Ketones I and II are converted to alcohols under biphenyl reduction for many chlorine-containing pesticides, and Lakshimarayana (1974) proposed the Beckman (1963) method of liberating origanically bound chlorine (refluxing sample in a hydrocarbon solvent with a boiling point above 100°C) with subsequent electrometric chlorine determination based on the procedure described by Northrup (1948).

The combustion or quartz tube method and the classical Parr bomb method initially used in composition analysis of endrin and other chlorinated pesticides are no longer considered definitive (Gunther and Blinn, 1955).

The total chlorine method as applied to technical endrin is a nonspecific method; that is, it determines all of the chlorinated compounds present in the sample. However, this technique is widely used as a plant control procedure for formulation of technical chlorinated pesticides or in situations where the pesticide formulations or extracts are devoid of any other chlorine-containing ingredients.

The spectrophotometric method used for the determination of endrin and its technical formulations is outlined in the Chemical Specialties Manufacturers Associations chemical analysis test methods (1972) and the AOAC methods book (1975). This method uses the absorption of endrin in the infrared region of the spectra and employs the absorption peak at 11.76 mm with a baseline drawn between 11.5 and 11.97 mm. The measurements are most commonly made in chloroform or carbon tetrachloride, and recommendations for the removal of any interfering absorption must be observed.

A relatively new technique which generates the mass spectra of a material, chemical ionization (CI) mass spectrometry, was described by Munson and Field (1966). The characteristic ionization spectra of the material in question are produced by ionic reactions rather than by electron impact (Damico et al., 1968). The ionic reactions take place in methane or mixtures of methane with small concentrations of other compounds at pressures up to 2 torr within the source of the mass spectrometer. The presence of small amounts of certain substances produces very marked changes in the relative concentration of the ions formed from the methane. These changes are the result of rapid reactions between the product ions of methane and the added material. The newly formed ions, in turn, produce a spectrum which is characteristic of each of the substances added.

The application of CI mass spectrometry as an analytical tool for the determination of chlorinated cyclodienes was reported by Biros et al. (1972) and Dougherty et al. (1972). These investigators, using methane as the reacting gas, were able to show that the CI mass spectra of endrin were less complex than the electron impact counterpart. The authors examined the positive and negative CI spectra of ten other polychlorinated cyclodienes and pointed out the usefulness of this technique for the determination of endrin and its degradation products. It was also suggested that because of the simplicity of the spectra, CI mass spectra could be applied to the direct examination of crude residue extracts for both identity and quality.

The application of mass spectrometry coupled with gas chromatography for endrin determination at the nanogram level was reported by Abrahamson (1972). Although the instrumentation used is often beyond the economic capabilities of many analytical laboratories, the technique offers (1) relatively uniform, high sensitivity for all materials which volatilize, (2) excellent selectivity from interfering materials (use of specific ion detector or high resolution), (3) freedom from false positive results, and (4) the ability to identify unexpected materials easily.

Colorimetric methods have been employed for residue rather than for composition analysis. However, Terriere (1964) described a method involving the reaction of endrin with phenyl azide to form a phenyldihydrotriazole derivative. The triazole is dissolved in ethanol and treated with hydrochloric acid. The resulting secondary amine is coupled with diazotized dinitroaniline to form an intense red color. The method is sensitive, but requires high dilution leading to a decrease in precision. Tewari and Sharma (1977) also reported a specific color reaction for endrin sensitive up to 0.05 mg endrin per 100 g of tissue.

Visweswariah and Jayaram (1971) described a semiquantitative colorimetric procedure using acetone solutions of endrin, among other chlorinated pesticides, hydrolyzed with monoethylamine and heat. The hydrolyzed solutions are then treated with silver nitrate to produce a white gelatinous turbid suspension characteristic of endrin. The procedure can be made quantitative by comparison with predetermined turbidity charts.

Thin-layer chromatographic (TLC) techniques are regarded as simple, quick, inexpensive, and easily reproducible. This method has been used for the determination of endrin in commercial formulation as well as for residue analysis. The procedure requires glass plates varying in size from 20 × 20 cm (Bontoyan and Jung, 1972) to microslides 2.5 to 7.5 cm (Visweswariah et al., 1971). The plates are usually covered with a pure silica gel or an aluminum oxide layer with or without silver nitrate (Visweswariah et al., 1971; Reinke et al., 1973; Bontoyan and Jung, 1972; Lakshimarayana and Menon, 1971; Sherma and Bloomer, 1977). Thin-layer chromatographic testing has been recommended as a confirmatory test or for the isolation of endrin prior to its GLC determination. The TLC procedure has also been used as a qualitative or quantitative analytical tool. The sensitivity of the method varies between 0.1 g (GLC) to 1.0 g endrin (visual).

The GLC methods used in the determination of endrin in commercial formulations and residue extracts lack some precision, but are rapid, simple, sensitive, and highly specific. The ability of the gas chromatographic procedures to measure extraordinarily low levels of endrin, from nanogram (10^{-9} g) quantities to picogram (10^{-12} g) quantities, has encouraged several scientists to look more closely to GLC methodology for the determination and quantification of endrin in biological specimens, environmental samples, and agricultural commodities.

In 1962, Phillips et al. developed a chromatographic technique using 710 silicone oil on GC-22 supersupport as a chromatographic column. The two peaks found at a column temperature of 270°C were believed to be the catalytic product of endrin isomerization due to impurities present in the analyzed extract or the lack of cleanup procedures. Direct GLC determinations of endrin in agricultural, atmospheric, and industrial samples were obtained by using electron-capture-ionization detectors. Goodwin et al. (1961) reported 95% recoveries from crop extracts (apples, broccoli, cabbage, carrots, lettuce, etc.) containing 0.1 to 0.25 ppm endrin. The quickness of the method, the elimination of sample cleanup, and the versatility of the electron-capture GLC technique were emphasized by these researchers.

Recognizing that the most vital component of the GLC technique is the chromatographic column itself, Chau and Wilkinson (1972a) found that a combination of OV-101 and OV-210 silicon oils on Chromosorb-W columns not only provided a means of separating photoendrin (half-cage ketone) from endrin but also retained good separation properties for the routine analysis of other organochlorine pesticides. The retention times of endrin and its photoproduct, using 2-1/2 SE-30 columns, were nearly identical (Zabik et al., 1971). Hastings and Aue (1974) studied the advantages of using chromatographic columns consisting of Carbowax 20-M modified celite covered with a 3% load of Apiezon L (Applied Science Laboratory, State College, Pennsylvania). The presence of an ultrathin, nonextractable film of Carbowax 20-M in a support used in typical GLC exerts a strong effect on the state of deactivation, but does not significantly influence the separation pattern prescribed by the liquid phase; furthermore, the quality of the chromatographic separation of aldrin, dieldrin, endrin, and heptachlorepoxide was much superior when Carbowax 20 modified celite columns rather than bare celite columns were used.

Burke and Holswade (1964, 1966) found that during the residue determinations of polycyclodiene insecticides by electron-capture GLC, interferences occurred not only from peak overlap of the various pesticides themselves, but also from extraneous contamination, for example, the laboratory, the technique, or naturally occurring components coextracted from the sample. The use of confirmatory procedures for the positive identification of endrin has been suggested by several investigators (Bowman and Beroza, 1965; Schutzmann et al., 1966; Fahey and Schechter, 1961; Bann et al., 1958; Woodham et al., 1972; Wiencke and Burke, 1969; Chau and Cochrane, 1969, 1971 among others). These procedures involve either of two well-known principles: (1) acid-catalyzed isomerization or (2) derivative formation by reacting endrin with an appropriate reagent.

Lidov et al. (1950) and Soloway et al. (1960) treated endrin with mineral or Lewis acids and obtained the half-cage pentacycloketone alkaline conditions, and the alcohols are silylated or acetylated. The GLC retention times of silyl esters or acetates are different from that of endrin. Thus, this procedure makes it possible to differentiate between unreacted endrin and its photodegradation products.

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3. BIOLOGICAL ASPECTS IN MICROORGANISMS

3.1 SUMMARY

Endrin is degraded by only a few microorganisms, all of which also degrade dieldrin. The metabolic pathways are speculative; however, degradation appears to begin on the nonchlorinated epoxy ring, resulting in the formation of ketones and aldehydes which may undergo dechlorination. Ketoendrin is the only metabolite which has been positively identified to date. Degradation of endrin does not always result in detoxification. Metabolites can be more toxic than the precursor and are often more stable. Degradation products can also stimulate growth where endrin exhibited an inhibitory effect.

Anaerobic conditions, such as those caused by flooding, and soils of high organic content increase the transformation of endrin by soil microorganisms (bacteria, fungi, and molds). In the marine environment, only samples from those areas having large algal populations are active in transformations.

3.2 BACTERIA

3.2.1 Metabolism

Relatively few bacteria are capable of degrading endrin. Only 2 to 17% of the microorganisms isolated from soil are active in endrin degradation (Matsumura et al., 1971; Brooks, 1974b). Endrin persists in soils for longer than nine months (Mulla, 1960, cited in Alexander, 1965b), implying that continued application will build up toxic levels to nontarget organisms. Little is known of the chemical, biochemical, and pedological bases for endrin resistance, but Alexander (1965b) suggests several factors that may be involved:

- inaccessibility of the endrin substrate (adsorbed or embedded),
- absence of a growth factor (carbon, nitrogen, etc.),
- toxicity of the environment (temperature, salinity, etc.),
- inactivation of the requisite enzyme,
- structural characteristics that prevent enzyme activity (branching molecule),
- physiological inadequacy (no enzyme to cope with the endrin substrate).

The generality that chlorine-containing rings are stable and that the major microbial attack is on nonchlorinated rings appears to hold true for endrin (Matsumura, 1973). Microorganisms rearrange the epoxy ring to form ketones, aldehydes, and alcohols (Fig. 3.1). While the ketone (ketoendrin) appears to be the dominant product of biotransformation and the only one identified to date, there are indications that reductive dechlorination of both aldehydes and ketone occurs (Patil et al., 1970; Matsumura et al., 1971; Brooks, 1974b).

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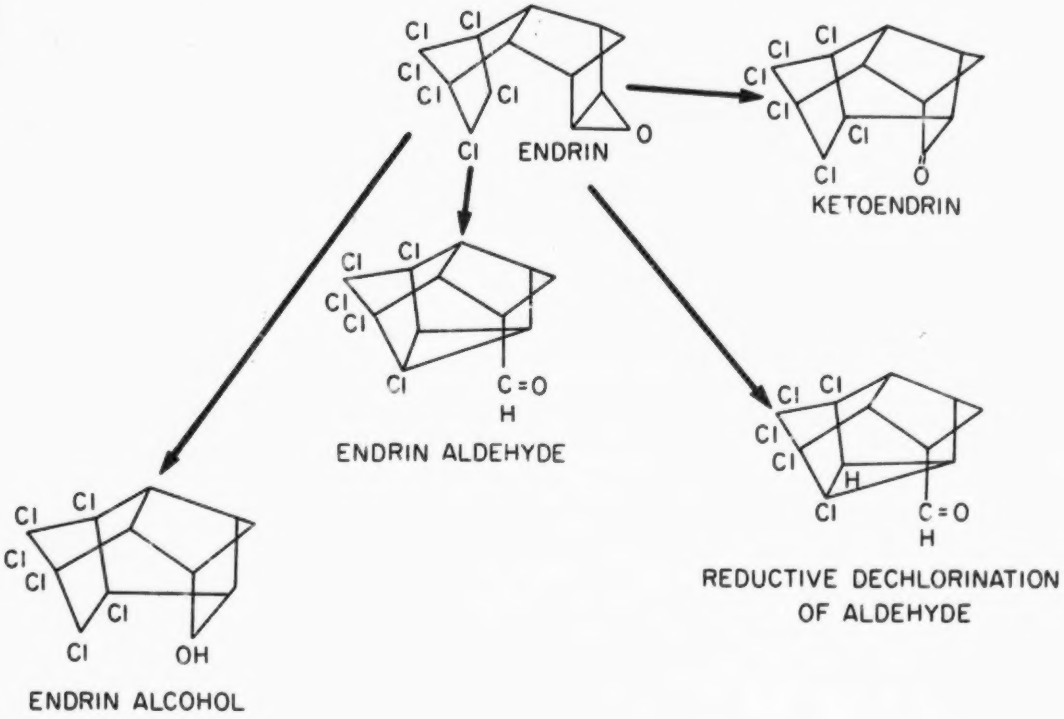

Fig. 3.1. Microbial degradation of endrin. Source: F. Matsumura, *Environmental Pollution* by Pesticides, 1973. Copyright 1973 by Plenum Publishing Corp.

All endrin-degrading bacteria were also found to degrade dieldrin (but not the reverse) (Patil et al., 1970). Seven metabolities of endrin (III, IIIa, IV, V, VI, and two others) were separated from soil cultures, of which only ketoendrin (IV) was identified (Table 3.1) (Matsumura et al., 1971). Ketoendrin ranged from undetectable to 46.3%, and the unaltered endrin ranged from 5.8 to 97.1% in the cultures. Biotransformation of endrin varied considerably, not only between different bacterial cultures but also between cultures of the same genus (e.g., *Pseudomonas spp.*). Patil et al. (1970) found similar results with soil isolates but separated only four endrin metabolites. The active

Table 3.1. Percent distribution of endrin and its solvent-extractable metabolites due to microorganisms (based on radioactivity)

Culture number	Microorganism (if identified)	Metabolite						
		0	III	IIIa	IV	V	VI	Endrin
17		2.0		2.7	3.4			91.9
18		4.6		4.2	3.2	6.7		81.3
19	Bacillus	2.0		2.1	4.6			91.3
20		1.4	10.4	27.9	19.3			41.0
26		4.9	1.9		3.4	44.9		44.9
44		2.2	20.6	5.9	19.5	32.2		19.6
48		1.1		2.5	3.3			93.1
49		3.2		2.7	3.3			90.8
52		2.1			16.0			81.9
53		0.5	13.3	19.0	29.4			37.8
83		1.2					18.5	80.3
93		0.7	36.5		9.5	38.0	9.5	5.8
95	Micrococcus	1.1	32.3		7.6	40.3		18.7
97		0.6	12.5		10.8	35.1		41.0
98		3.6	6.8		9.4	15.3		64.9
100		0.6			2.3			97.1
103	Pseudomonas	0.5	13.0	Trace	46.3	16.2	9.0	15.0
115		0.6			3.1			96.3
117	Pseudomonas	1.4			16.1			82.5
118		1.4			11.0			87.6
119		0.8			5.8			93.4
120		2.1			9.6			88.3
136	Pseudomonas	1.9			9.8	10.1		78.2
137		1.3	5.8		20.2	14.8		57.9
(173	Yeast	4.1			80.6			15.3)

Source: F. Matsumura et al., *J. Agric. Food Chem.* 19(1): 27-31 (1971). Copyright 1971 by the American Chemical Society.

Brooks (1974b) postulates that an endrin metabolite, if heated, would lose CO to give a derivative of hexachlorobenzonorbornene. If both bridges were removed by biological degradation, derivatives of tetrachloronaphthalene would result. Microorganisms appear capable of genrating olefins from epoxides (i.e., dieldrin to aldrin), or molecules may be modified to structures more vulnerable to conversion into water-soluble compounds (conjugates), by reacting with endogenous chemicals through enzyme catalyzation (Brooks, 1974b). For example, bacteria form glucosides by attaching endogenous glucose with molecules such as alcohols in this way.

bacteria included *Pseudomonas spp.*, *Bacillus spp.*, *Trichoderma spp.*, *Micrococcus sp.*, *Arthrobacter sp.*, as well as unidentified cultures. *Corynebacterium pyogenes* appeared to decrease liquid endrin concentrations in 7 to 16 days, but two species of *Pseudomonas* (*P. fluorescens* and *P. aeruginosa*) were without effect (Cope, 1963).

Endrin is very persistent under both aerobic and anaerobic conditions (Pfister, 1972), but the creation of anaerobic conditions generally stimulates microbial degradation of endrin. Support for this phenomenon includes increasing transformation of endrin with increasing depth in field soil (Nash et al., 1972) and decreasing endrin in flooded soil compared with upland conditions (Castro and Yoshida, 1971; Guenzi et al., 1971). Endrin degradation is not affected by temperature (Pfister, 1972) or soil mixing, but it is improved by the amount of organic material in flooded or naturally occurring soils (Table 3.2) (Guenzi et al., 1971; Castro and Yoshida, 1971). Bacterial involvement was suggested by organic enhancement (i.e., as an energy source) and by the apparent lag time in endrin disappearance (i.e., adaptation time).

Table 3.2. Concentration of endrin under different soil conditions (ppm)

Weeks	2.5 cm water/week		2.5 cm water/week, soil mixed		Flooded	
		+ Alfalfa		+ Alfalfa		+ Alfalfa
0	25	25	25	25	25	25
2	20.4	21.9	20.0	20.5	19.4	21.7
6	19.5	20.5	20.9	19.3	18.6	19.5
10	19.8	19.5	20.3	20.2	17.1	15.2
14	20.1	19.9	20.7	19.4	15.5	12.7
18	20.5	21.0	18.9	20.7	14.1	10.8
22	19.2	19.9	19.1	19.9	14.6	11.0

Source: W. D. Guenzi et al., *Soil Sci. Soc. Am. Proc.* 35(6): 910-913 (1971). Copyright 1971 by the Soil Science Society of America.

Since the metabolic pathways of endrin in microorganisms are speculative at this time, discussion of similar transformations may help elucidate endrin reactions. All of the endrin metabolites classified so far have the hexachlorinated norbornene moiety unchanged. However, under anaerobic conditions, *Clostridium butyricum* reduced the dichloromethanobridge in hexachloronorbornene (compound 1, Fig. 3.2) to $-CH_2$ (compound 2, Fig. 3.2) and further hydroxylated it to -CH(OH)- (compound 3, Fig. 3.2). A tetrachlorocyclodiene (compound 4, Fig. 3.2) was also found in the bacterial culture, apparently formed only from the fully chlorinated molecule by direct dechlorination without intermediate alcohol formation (Schuphan and Ballschmiter, 1972).

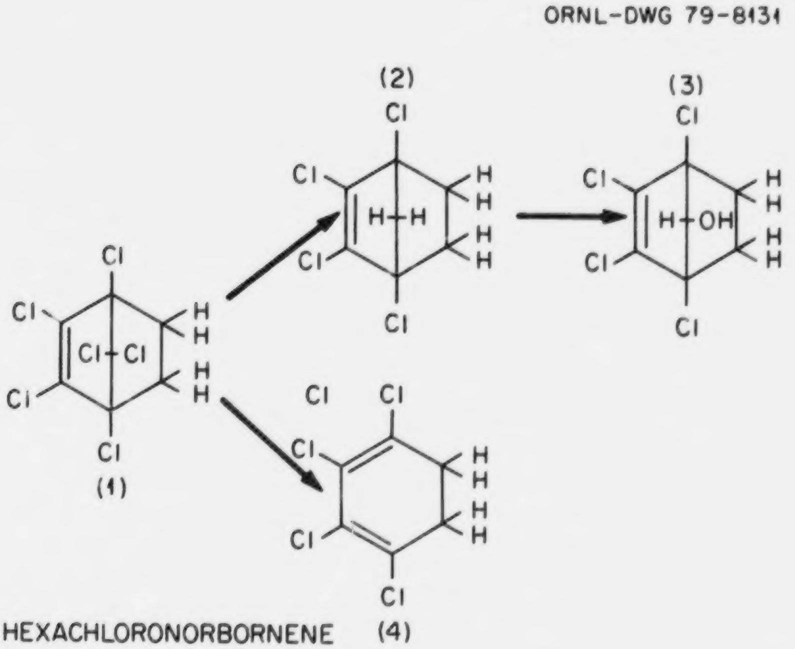

Fig. 3.2. Anaerobic transformations of hexachloronorbornene by *Clostridium butyricum*. Source: I. Shupman and K. Ballschmiter, *Nature* 237: 100-101 (1972). Copyright 1972 by MacMillan Journals Ltd., London.

No dechlorinated products of hexachloronorbornene were found in yeast (*Saccharomyces*) cultures or with soil microorganisms (Schuphan and Ballschmiter, 1972). The extent to which these reactions can be generalized for endrin is unknown; however, hypothetical transformations involving oxidation (hydroxylation), oxidative dechlorination, reductive dechlorination, hydrochlorination, hydrolytic removal of chlorine, hydration of epoxide rings, and skeletal rearrangements seem more probable in view of similar reactions that are known.

The significance of the rearrangements is not known, but Matsumura (1973) believes that it is unlikely that microbes derive any energy. Usually biotransformation generates more polar and degradable products than do the parent compounds, so rearrangements may provide further opportunities to break down the molecules (Brooks, 1974b; Matsumura, 1973). It has also been suggested that anaerobic (reductive) dechlorination of endrin by microorganisms is precursory to aerobic (oxidative) degradation, as indicated for DDT (Brooks, 1974b). Thus, one way organic matter may aid degradation of endrin is by decreasing the oxidation-reduction potential of the soil, thereby creating conditions favorable to reductive dechlorination (Castro and Yoshida, 1971).

The identification of "terminal residues" is important, because biotransformation products may not follow the same pathways as do precursor molecules. Metabolic products may be eliminated on the one hand or cause toxicity not demonstrated by parent compounds on the other hand. Thus, different mechanisms of activation, transformation, and detoxification are bases for variation on organism sensitivity to endrin.

3.2.2 Effects

Microorganisms that fix and release nitrogen are essential to the biosphere, providing the major means of introducing elemental nitrogen into organic compounds. The toxicity of endrin to these soil microorganisms is vitally important, but Pfister (1972) and Bollag and Henninger (1976) reported that endrin was not excessively inhibitory to ammonifying organisms or decomposers that liberated ammonia. In fact, Eno and Everett (1958) conclude that pesticides appear to be toxic to higher plants (12.5 ppm) before microbial responses are observed. The lack of effects probably is explained by low solubility in water and low vapor pressure, limiting the amount of chemical in soil solution and, therefore, the quantity contacting microorganisms.

Many soil fungi (including Streptomyces, Penicillia, and molds) and bacteria are not affected at concentrations below 1000 ppm (Pfister, 1972; Bollen and Tu, 1971). Endrin concentrations up to 50 mg/liter had no toxic effect on sewage organisms for five days, based on BOD and COD studies (Canter et al., 1969). *Escherichia coli* cultures grew for 24 hr on agar and in suspension with 500 and 1500 mg/liter endrin added, respectively, and complete growth inhibition occurred at 3000 mg/liter (Canter et al., 1969). The decomposition rate of soil organic matter, as measured by CO_2 evolution, was the same in treated and control samples, as was sulfur oxidation and ammonification below 1000 ppm and nitrification below 100 ppm (Bollen and Tu, 1971). Endrin was found to stimulate degradation of organic matter in situ at levels below inhibition, but in the laboratory, microbial plates were not affected (Bollen and Tu, 1971).

Bacterial sensitivity to endrin appears to be related to their gram response (Trudgill et al., 1971). Gram-negative and -variable bacteria were not affected by surface films of 0.5 mg/ml endrin, but 50% of the gram-positive cultures were inhibited (Table 3.3). While some discrepancy occurs in the effect of endrin on the growth of *Bacillus* species (Patil et al., 1970; Trudgill et al., 1971), differences in methods of growth determination and incubation periods may be responsible.

3.3 PROTOZOA AND PLANKTON

Conversions of chemical compounds in the natural environment may result in more stable or sometimes more toxic products than the parent compound. A degradation product of endrin, ketoendrin, produced by the blue-green alga *Anacystis nidulans*, proved to inhibit growth as much as the parent compound (Fig. 3.3) (Batterton et al., 1971). *Agmenellum quadruplicatum,* another blue-green alga, was slightly stimulated by ketoendrin, while inhibited by endrin. Thus, not all degradation products represent a detoxification step. *A. nidulans* had a lag period of up to

Table 3.3. Effects of endrin on bacterial growth

Gram positive	
Bacillus megaterium	-
B. subtilis	-
Streptomyces antibioticus	-
Nocardia sp. B	-
Corynebacterium sp. T I	-
B. cereus	-
Nocardia sp. A	+
Microbacterium flavum	+
Micrococcus lysodeilticus	+
Staphylococcus aureus	+
Sarcina lutea	+
Gram variable	
Arthrobacter simplex	+
Gram negative	
Achromobacter butyri	+
Achromobacter sp. PC$_4$	+
Escherichia coli	+
Klebsiella aerogenes	+
P. acidovorans	+
P. aureofaciens	(+)
P. dehalogens	+
P. fluorescens	+
P. fluorescens	+
P. multivorans	+
P. putida	+

+, growth not inhibited; (+), growth slightly inhibited; -, growth severely or completely inhibited.

Source: P. W. Trudgill et al., *J. Gen. Microbiol.* 69(1): 1-13. Copyright 1971 by the Cambridge Univ. Press.

12 hr in the presence of 1 ppb endrin concentration during which apparently adaptation or selection of resistant cells took place (Batterton et al., 1971; Matsumura, 1972).

Mixed algal cultures isolated from fish-pond water and the fish-pond water per se degraded radiocarbon-labeled endrin (Patil et al., 1972). Of the introduced carbon label, 24.4% was recovered in ketoendrin from algal cultures, and 35.5% from the pond water was in an unknown metabolite.

Some blue-green algae have the capability of fixing elemental nitrogen into organic compounds, thereby providing nitrogen in a usuable form to other organisms. The effects of endrin on two cultures of these ecologically significant blue-greens were reported by Singh (1973). Both

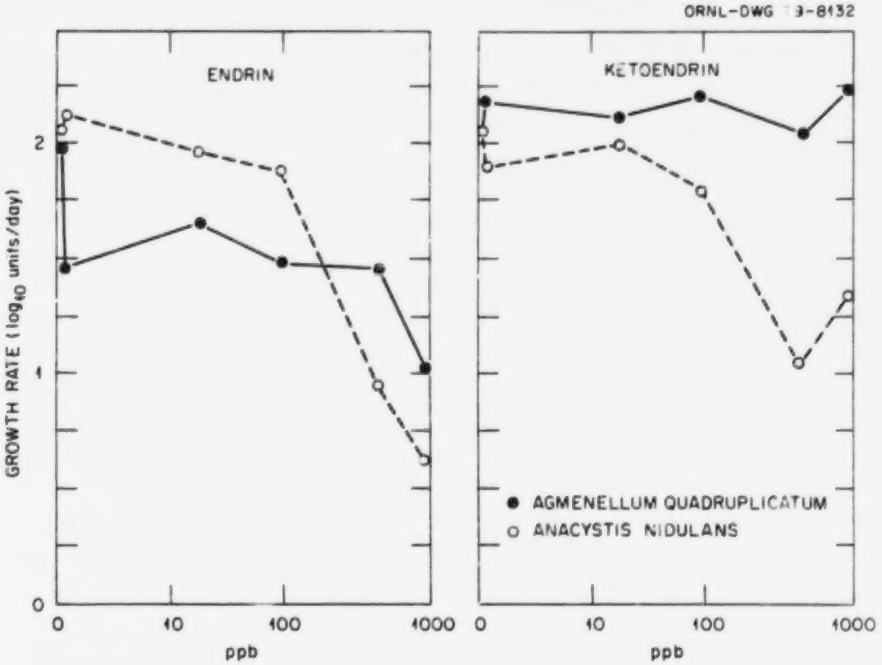

Fig. 3.3. Effect of endrin and one of its degradation products, ketoendrin, on two species of blue-green algae, after 36 hr. Source: J. C. Batterton, G. M. Boush, and F. Matsumura, *Bull. Environ. Contam. Toxicol.* 6(6): 589-594 (1971). Copyright 1971 Springer-Verlag, N.Y.

Cylindrospermum sp. and *Aulosira fertilissima* showed increasing growth inhibition with increasing endrin concentrations (Fig. 3.4), while an aerobic, non-nitrogen-fixing blue-green alga (*Plectonema boryanum*) was only slightly inhibited at 600 μg/mm endrin. No effect was observed on morphology or sporulation of nitrogen-fixing algae by endrin. However, heterocyst frequency was directly correlated with endrin concentration.

Cultures of four marine phytoplankton (*Skeletonema costatum*, *Dunaliella tertiolecta*, *Coccolithus huxleyi*, and *Cyclotella nana*) showed varying responses to endrin (Menzel et al., 1970; Pfister, 1972). *S. costatum* and *C. huxleyi* had radiocarbon uptake and cell division reduced at 10 and 100 ppb endrin respectively (Fig. 3.5). Cell division returned to normal after five days in *S. costatum*. *C. nana* proved most sensitive; radiocarbon uptake and cell division were completely inhibited above 1 ppb, with the dose-response curve suggesting effective doses as low as 0.01 ppb endrin (Menzel et al., 1970). There was no effect on *D. tertiolecta* with endrin concentrations up to 1000 ppb.

Algae concentrated endrin manyfold, either through ad- or absorption (Turnipseed, 1974; Vance and Drummond, 1969; Pfister, 1972). Concentration factors ranged from 140 to 222 for seven days, with slightly higher bioaccumulation in two blue-green algae, *Microcystis aeruginosa* and *Anabaena*

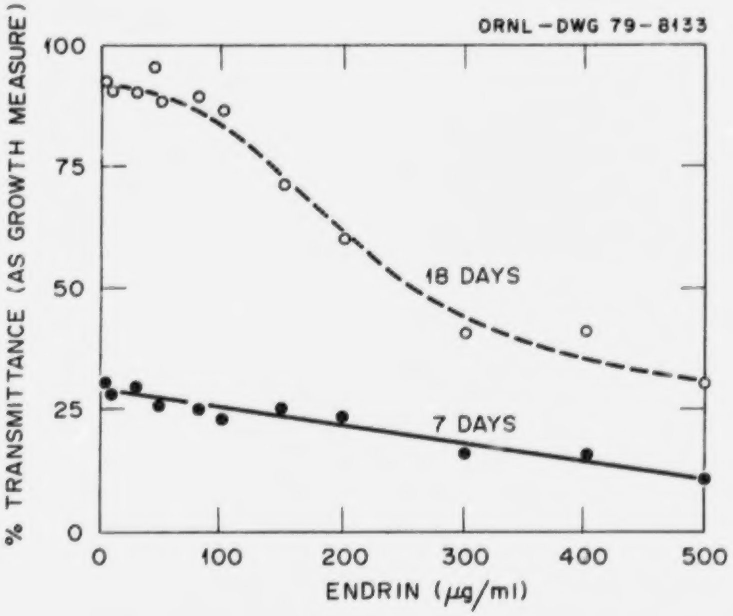

Fig. 3.4. Effect of endrin on *Cylindrospermum* sp. Source: P. K. Singh, *Arch. Mikrobiol.* 89(4): 317-320 (1973). Copyright 1973 Springer-Verlag, N.Y.

cylindrica, compared with two greens, *Scenedesmus quadricauda* and *Oedogonium spp.* In contrast, lethal concentrations were higher for the greens (>20 ppm) than for the blue-greens (<20 ppm) (Vance and Drummond, 1969). Bioconcentration increased in algae with decreasing exposure levels, although tissue accumulation per se was lower. For example, *Oedogonium spp.* had bioconcentration factors of 140 and 4500 associated with tissue levels of 140 and 11.6 ppm respectively (Metcalf et al., 1973; Vance and Drummond, 1969). *Chlamydomonas reinhardtii* had growth, respiration, and photosynthesis inhibited to some degree by endrin, but concentration levels were not given (Turnipseed, 1974).

Because of the low solubility of endrin, it was difficult to determine incorporation into cell material (Pfister, 1972); however, the response of some algae to concentrations above the solubility limits (about 0.2 ppm) suggested that either saturation was maintained while endrin was accumulated from solution or that there might be incorporation as small particulates. This last pathway is supported by the association of endrin with microscopic particles (0.15 μm or more) in Lake Erie water, including organics, detritus, and microorganisms (Pfister et al., 1969).

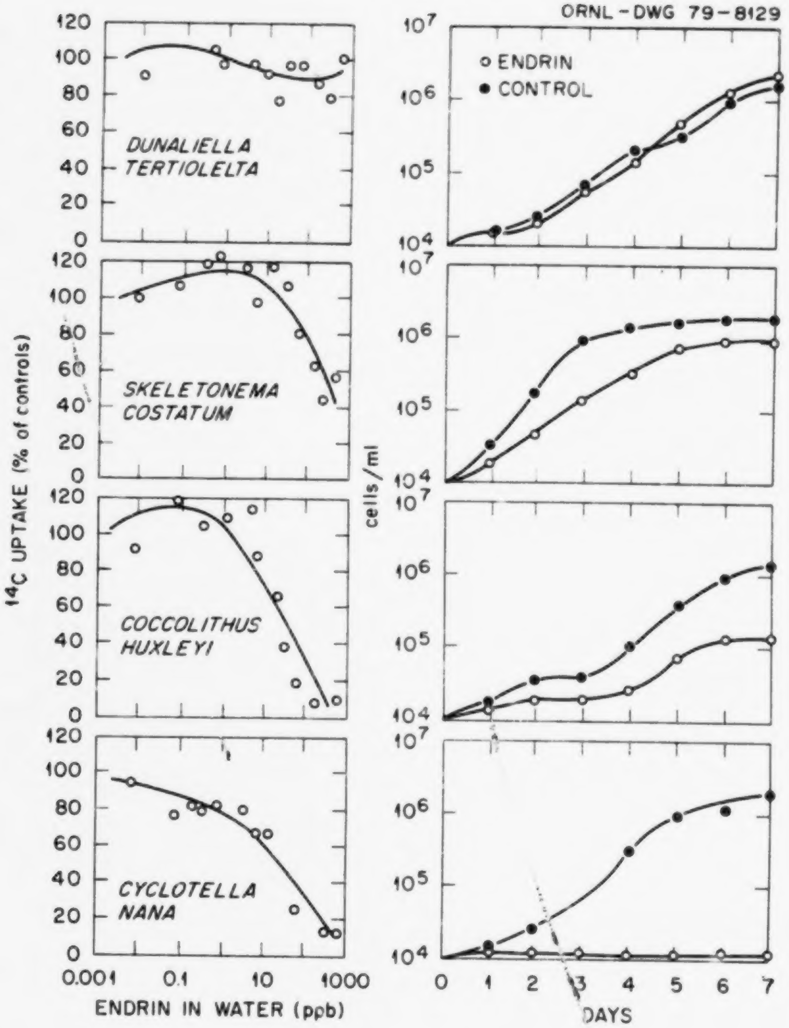

Fig. 3.5. Uptake of endrin by phytoplankton at various concentrations over 24 hr (left side) and growth rates of same species in 100 ppb endrin over seven days (right side). Source: D. W. Menzel, J. Anderson, and A. Randike, *Science* 167(3926): 1724-1726 (1970). Copyright 1970 American Society for the Advancement of Science.

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4. BIOLOGICAL ASPECTS IN PLANTS

4.1 SUMMARY

The fungi *Aspergillus niger*, *A. flavus*, *Penicillium notatum*, and *P. chrysogenum* metabilize endrin into ketoendrin and a very hydrophilic compound that are found in both mycelium and culture medium. The age of the culture affects endrin metabolism. In growing cultures of *A. flavus*, endrin was absorbed nearly quantitatively from the culture medium and accumulated in the mycelium, while the endrin metabolites (produced by a 2 to 3% metabolic rate) were excreted into the medium quantitatively. In full-grown cultures of *A. flavus*, the metabolic rate was 22%, and the endrin metabolites were found in both mycelium (20%) and culture medium (74%).

An unidentified yeast metabolized endrin at the metabolic rate of 84.1%; ketoendrin was the only metabolite.

Endrin inhibits cell growth of the yeast *Saccharomyces cerevisiae*. Inhibition was greater (17%) with the fermentable sugar glucose as substrate than it was (6%) when the culture medium contained nonfermentable lactate.

Uptake of endrin by vascular plants has been demonstrated. Plants grown in soil that contains endrin absorb it by the roots. Endrin applied to plant leaves is subsequently absorbed by the leaves. Leaves also absorb endrin vapors (e.g., endrin that has volatilized from the soil).

The amount of endrin taken up by vascular plants from soil depends on the plant, on the type of soil, and on the endrin concentration in the soil.

When they are grown under the same conditions, some plants develop higher endrin residues (concentration based on fresh or dry weight) than others. For example, carrot (*Daucus carota*) had higher concentrations of endrin than did other root crops, including potato (*Solanum tuberosum*), radish (*Raphonus sativus*), turnip (*Brassica rapa*), and beet (*Beta vulgaris*). In another experiment, endrin concentrations in aerial plant parts decreased in the order: bromegrass (*Bromus inermis*), alfalfa (*Medicago sativa*), wheat (*Triticum aestivum*), corn (*Zea mays*), and soybean (*Glycine max*).

Soil type also affects endrin uptake from soil. When plants were grown in different soils, it was found that endrin residues in aerial plant parts generally varied by a factor of 2, although the difference was sometimes as great as fivefold. Residues were usually highest in plants grown in sandy loam, and somewhat lower when the soil was clay or loam. Silt in the soil retarded endrin uptake.

Endrin uptake by plants appears to be proportional to the concentration of endrin in soil, at least at low concentrations. Endrin residues in plants grown in soil that contained 5.0 ppm endrin were tenfold the residues in plants grown in soil with 0.5 ppm endrin.

The absorbed endrin is translocated throughout the plant. Endrin concentrations in the various plant parts decrease with distance from the site of uptake. When ^{14}C-endrin was applied to and absorbed by leaves of cotton (*Gossypium hirsutum*) and cabbage (*Brassica oleracea capitata*), most of the recovered radioactivity was associated with the leaves. In cotton, radioactivity was also detected in stalks, pods, fibers, and seeds, but not in roots. In cabbage, recovered radioactivity was also associated with the stems and roots. Substantial amounts of radioactivity (20% with cotton and 9.2% with cabbage) were recovered in the soil in the translocated endrin and its metabolites that had been excreted by the roots.

Absorbed endrin is metabolized by plants, and as many as five metabolites have been detected. Three of the metabolites (including ketoendrin) are slightly more hydrophilic than endrin, and two are very hydrophilic. Delta-ketoendrin, the only identified metabolite, was detected by most investigators, sometimes as the only metabolite and sometimes as a minor constituent of the metabolite fraction, with the major constituent(s) being very hydrophilic. Endrin alcohol and traces of endrin aldehyde in plants have also been reported.

The rate of metabolism of endrin by vascular plants varies with the plant species and probably also with the mode of endrin application. The metabolic rate, as indicated by the proportion of metabolites in the recovered endrin residues, was about 10% in cabbage four weeks after foliar application of endrin at different rates. In tobacco (*Nicotiana tabacum*), the metabolic rate was only 2.3% after four weeks. In carrot, the metabolic rate three weeks after treatment with endrin was 32% with foliar application, but 12% when endrin was injected into the roots and 5% when endrin was applied to the soil.

The amount of radioactivity recovered, when ^{14}C-endrin was used in experiments, varied greatly. Recovery was always less than 50% four weeks after endrin application. Because it is known that endrin and its metabolites are excreted into soil by plant roots, most experiments of this type did include soil analysis. The discrepancy between the amounts of radioactivity applied and recovered has been attributed to vaporization of the applied endrin and also to loss of absorbed endrin and/or its metabolites by transpiration.

Endrin is somewhat phytotoxic to vascular plants. With the application of a 0.03% endrin emulsion, scorching of the foliage of three varieties of cucurbits was apparent to different degrees after seven days of growth. A 0.04% emulsion effected high phytotoxicity within three days of application. There are also reports that endrin is toxic to corn. Some adverse effects attributed to endrin are actually caused by the solvent used in the formulation.

Endrin at 1 ppm did not affect the rates of germination and growth of onion (*Allium cepa*). However, the germinability of barley (*Hordeum vulgare*) seeds soaked in endrin solutions at 500 and 1500 ppm for 24 hr was reduced 50%.

Mitosis in seedling root cells was affected by endrin treatment. A slight stickiness of anaphase chromosomes 48 hr after germination and an increase in the number of anaphases with incompletely separated chromatids ten days after germination were observed in onion. Endrin treatment induced chromosome aberrations in mitotic root cells of seedlings at mean frequencies of 7.59 and 18.55% in barley and broad bean (*Vicia faba*) respectively.

Meiosis in barley was not affected when barley seeds were soaked in a 1000-ppm solution of endrin for 12 hr prior to germination. However, when endrin at 500 ppm was sprayed onto 16-day-old seedlings, the frequency of chromosomal abnormalities in pollen mother cells increased about 3.5-fold.

The presence of endrin in the soil affects the uptake of various macro- and microelements by vascular plants. The uptake of some elements is increased, whereas the uptake of other elements is decreased. The actual effect is a factor of the particular element, the plant species, and the concentration of endrin.

The presence of endrin in soil alters the amino acid composition of plants. There were significant increases and decreases in the contents of various amino acids in corn grown in endrin-treated soil.

4.2 NONVASCULAR PLANTS

4.2.1 Metabolism of Endrin

Korte (1967) mentioned that the fungi *Aspergilles niger*, *A. flavus*, *Penicillium notatum*, and *P. chrysogenum* metabolize endrin. Metabolites were isolated from both the mycelium and the culture medium; they were more hydrophilic than endrin.

The experiments with *Aspergillus flavus* were later described in more detail (Klein et al., 1968b). Carbon-14-endrin was applied at concentrations of 0.044 to 0.132 ppm to cultures of *A. flavus*. Three weeks later, the mycelium and the culture medium were analyzed separately. Twenty-five percent of the applied radioactivity was not recovered. The 3% of applied radioactivity found in the cotton plugs of the culture flasks was associated only with endrin. It was concluded, therefore, that volatilization of endrin accounted for the lost radioactivity and that no metabolites were lost by volatilization. When the endrin was applied to growing cultures of *A. flavus*, it was absorbed nearly quantitatively and was accumulated by the mycelium. The metabolic rate was 2 to 3%, and the metabolites were excreted quantitatively into the culture medium. When the endrin was applied to full-grown (i.e., not growing) cultures, 20% of the metabolites were found in the mycelium and 74% in the culture medium; the metabolic rate was 22%. Two metabolites were produced by *A. flavus* — ketoendrin and a very hydrophilic compound; these metabolites were identical with the endrin metabolites of higher plants.

A yeast-mannitol medium was used to culture an unidentified yeast that had been isolated from farm soil (Matsumura et al., 1971). Ten microliters of 0.001 *M* ^{14}C-endrin solution was added to each culture test tube. Of the total recovered radioactivity (21% of the applied radioactivity), 80.6% was associated with ketoendrin and 15.3% with endrin; ketoendrin was the only metabolite.

4.2.2 Effects of Endrin

The effect of endrin on growth of the yeast *Saccharomyces cerevisiae* was examined by Nelson and Williams (1971). The liquid culture medium contained either 1% glucose or 2% lactate as the energy source. Immediately after the cultures were inoculated, 1 ml of 0.01 *M* endrin solution in dimethylsulfoxide was added to the 100 ml of medium in each flask. After a 20-hr incubation period, it was observed that cell growth had been inhibited 17% in the glucose-containing culture medium, but only 6% in the lactate-containing medium. This finding was surprising because the cyclodiene pesticides chlordane and heptachlor completely inhibited cell growth of *S. cerevisiae* when the substrate was nonfermentable lactate, and other cyclodiene pesticides (heptachlor epoxide, aldrin, and dieldrin) also strongly inhibited cell growth in lactate-containing medium. When the energy source was the fermentable sugar glucose, cell growth was inhibited 13 to 20% by the various cyclodiene pesticides (37% by chlordane).

Chlorinated hydrocarbons, including endrin, inhibit fermentation processes in *Aspergillus niger* and *Saccharomyces cerevisiae* (Singh et al., 1977). Production of citric acid by *A. niger* was reduced 40 to 60% compared with the control. A similar inhibition pattern was also observed on fermentation of ethyl alcohol by *S. cerevisiae*. Initial steps of fermentation do not seem to be affected by chlorinated hydrocarbons, since sugar utilization is not inhibited by these compounds.

4.3 VASCULAR PLANTS

4.3.1 Metabolism of Endrin

Most of the earlier studies of endrin and plants were confined primiarily to quantifying endrin residues (e.g., Brett and Bowery, 1958;

Bowery et al., 1959; Gyrisco and Huddleston, 1961; Mattick et al., 1963; Archer, 1968b; Bhalla et al., 1970; and Knutson et al., 1971). The investigators did not, and often could not, distinguish between the residues on the plant surfaces and those within the plant tissues.

More sophisticated experiments were designed to determine whether endrin is absorbed into plants, translocated within the plants, and metabolized by the plant. These experiments established that plants do absorb, translocate, and metabolize endrin.

In order to demonstrate root uptake of endrin and its translocation, plants were grown in soil which contained endrin that had been applied to the surface of the soil or mixed in with the top soil layer. No endrin was sprayed on the plants, and, therefore, the only source of endrin was that present in the soil.

Although the quantity of endrin absorbed from the soil is affected by several factors, root uptake of endrin does occur (Table 4.1). It has been reported for many root crops (Foster et al., 1956; Harris and Sans, 1967; Hermanson et al., 1970; Korte, 1967; Van Middelem, 1969b; Wheeler et al., 1969; Winnett and Reed, 1968) as well as for diverse plant types, including grasses (Beall and Nash, 1969; Saha and co-workers, 1967, 1968), legumes (Barrentine and Cain, 1969; Beall and Nash, 1969, 1971; Nash and co-workers, 1970, 1971, 1972, 1973, 1974b; Saha et al., 1968; Van Middelem, 1969b), cucurbits (Beall and Nash, 1969), and pomes (Horsfall et al., 1970).

Working with turnips (*Brassica rapa*) and carrots (*Daucus carota*), respectively, Wheeler et al. (1969) and Hermanson et al. (1970) proved that endrin residues in root crops could not be attributed only to surface contamination. The presence of endrin in peeled roots indicates that the insecticide is absorbed from the soil.

Other investigators reported the presence of endrin residues in aerial parts of plants, including the leaves of bromegrass (*Bromus inermis*) and alfalfa (*Medicago sativa*) (Beall and Nash, 1969), sweet clover (*Melilotus* sp.), oat (*Avena sativa*), barley (*Hordeum vulgare*) (Saha et al., 1968), wheat (*Triticum aestivum*) (Beall and Nash, 1969; Saha and co-workers, 1967, 1968), turnip (Van Middelem, 1969b; Wheeler et al., 1969), and tobacco (*Nicotiana tabacum*) (Van Middelem, 1969b). Endrin residues were also detected in soybeans (*Glycine max*) (Barrentine and Cain, 1969; Beall and Nash, 1971; Nash and co-workers, 1970, 1973, 1974b) and in apples (*Malus domestica*) (Horsfall et al., 1970). These findings evidence translocation of endrin within the plant subsequent to its absorption by the root.

The amount of endrin absorbed from the soil depends on various factors, including plant species, insecticide concentration, and soil type. Hermanson et al. (1970) noted that carrots usually take up more endrin than do other root corps [e.g., potato (*Solanum toberosum*), radish (*Raphonus sativus*), turnip, and beet (*Beta vulgaris*)]. Beall and Nash (1969) grew soybean, wheat, corn (*Zea Mays*), alfalfa, bromegrass,

Table 4.1. Absorption of endrin from soil by plants

Plant	Treatment		Endrin in soil[a] (ppm)	Growth period	Endrin residues			Reference
	(lb/acre)	(ppm)			Plant part	Compound	Concentration[b] (ppm)	
Soybean	2		0.59	5 months	Beans	Endrin	0.04 (F)	Barrentine and Cain, 1969
	4		1.20	5 months	Beans	Endrin	0.08 (F)	
	8		1.49	5 months	Beans	Endrin	0.12 (F)	
Soybean		0.5		3 weeks	Above cotyledon	Endrin	0.024–0.045 (D)	Beall and Nash, 1969
		5.0		3 weeks	Above cotyledon	Endrin	0.303–0.410 (D)	
Wheat		0.5		3 weeks	Above coleoptile sheath	Endrin	0.176 (D)	
		5.0		3 weeks	Above coleoptile sheath	Endrin	1.844 (D)	
Corn		0.5		3 weeks	Above coleoptile sheath	Endrin	0.068 (D)	
		5.0		3 weeks	Above coleoptile sheath	Endrin	0.708 (D)	
Alfalfa		0.5		3 weeks	Above cotyledon	Endrin	0.215 (D)	
		5.0		3 weeks	Above cotyledon	Endrin	2.464 (D)	
Bromegrass		0.5		3 weeks	Above coleoptile sheath	Endrin	0.304 (D)	
				6 weeks	Above coleoptile sheath	Endrin	0.451 (D)	
		5.0		3 weeks	Above coleoptile sheath	Endrin	3.127 (D)	
				6 weeks	Above coleoptile sheath	Endrin	4.514 (D)	
Cucumber		0.5		4 weeks	Above cotyledon	Endrin	0.028 (D)	
		5.0		4 weeks	Above cotyledon	Endrin	1.084 (D)	
Soybean		20	20	53 days	Above cotyledon	Endrin + 2 metabolites	38.1 (D)	Beall and Nash, 1971
					Leaves		125.95 (D)	
					Stem		303.93 (D)	
					Pods		10.44 (D)	
					Seeds		3.30 (D)	
Carrot		15	15.8	11.5 weeks	Roots	Total organic chlorine	4.0 (F)	Foster et al., 1956
		21.7	16.9	13 weeks	Roots	Total organic chlorine	3.0 (F)	
Sweet potato			4.4	23 weeks	Roots	Total organic chlorine	7.3 (F)	
Carrot			8.91	14 weeks	Whole plant	Endrin	0.06 (F)	Harris and Sans, 1967
Raddish			8.91	5 weeks	Whole plant	Endrin	0.04 (F)	
Sugar beet	5				Roots	Endrin	Significant	Hermanson et al., 1970

Table 4.1 (continued)

Plant	Treatment		Endrin in soil[a] (ppm)	Growth period	Endrin residues			Reference
	(lb/acre)	(ppm)			Plant part	Compound	Concentration[b] (ppm)	
Carrot	14		3.6-3.8	142 days	Roots	Total organic chlorine	1.2-1.6 (F)	
					Peeled roots	Total organic chlorine	0.5-0.7 (F)	
					Roots	Endrin	1.2-4.0 (F)	
				163 days	Roots	Endrin	1.0-3.2 (F)	
Apple	2			11 months[c]	Fruit	Endrin	<0.0005-0.005 (F)	Horsfall et al., 1970
	2			6 months[c]	Fruit	Endrin	<0.002-0.028 (F)	
	4			[d]	Fruit	Endrin	<0.002-0.007 (F)	
	4			6 months[c]	Fruit	Endrin	<0.002-0.023 (F)	
Carrot			0.04	3 weeks	Roots	Endrin + metabolite	(Uptake, 26%)	Korte, 1967
Soybean			20	53 days	Leaves	Endrin + metabolite(s)	(19.0% endrin)	Nash and Beall, 1970
					Stems	Endrin + metabolite(s)	(57.3% endrin)	
					Seeds	Endrin + metabolite(s)	(2.7% endrin)	
Soybean			20	53 days	Stems and leaves	Endrin + 2 metabolites		Nash and Beall, 1971
Soybean			20	75 days	Above cotyledon	Endrin + 3 metabolites		Nash et al., 1972
Soybean			7.52	99 days	Beans	Endrin + 3 metabolites	0.096 (F)	Nash and Harris, 1973
				118 days	Beans	Endrin + 2 metabolites	0.032 (F)	
			21.44	99 days	Beans	Endrin + 2 metabolites	0.108 (F)	
				118 days	Beans	Endrin + 3 metabolites	0.098 (F)	
Soybean			20	43 days	Aerial	Endrin + metabolites	0.266	Nash, 1974b
				99 days	Aerial	Endrin + metabolites	0.202	
				160 days	Hay	Endrin + metabolites	0.195	
				160 days	Seed	Endrin metabolites	0.026	
Wheat	2		1.00-1.12	45 days	Plants	Endrin	0.009-0.021 (F)	Saha and McDonald, 1967
				60 days	Plants	Endrin	0.014-0.020 (F)	
				112 days	Straw	Endrin	0.022-0.028 (F)	
	8		3.65-3.76	45 days	Plants	Endrin	0.040-0.050 (F)	
				60 days	Plants	Endrin	0.068-0.082 (F)	
				112 days	Straw	Endrin	0.046-0.054 (F)	
Sweet clover	8		1.20		Aerial	Endrin	0.1[illegible] (F)	Saha et al., 1968
Wheat	8		1.20		Aerial	Endrin	0.03-0.05 (F)	Saha et al., 1968
Oat	8		1.20		Aerial	Endrin	0.03-0.05 (F)	
Barley	8		1.20		Aerial	Endrin	0.03-0.05 (F)	
Soybean	8	4			Beans	Endrin	0.02-0.11	Van Middelem, 1969b

Table 4.1 (continued)

Plant	Treatment		Endrin in soil[a] (ppm)	Growth period	Endrin residues			Reference
	(lb/acre)	(ppm)			Plant part	Compound	Concentration[b] (ppm)	
Turnip	4	2			Greens	Endrin	Insignificant	
					Root peels	Endrin	0.12	
Tobacco	4	2			Green leaves	Endrin	0.04	
Turnip	1		0.64	66 days	Greens	Endrin	0.01 (F)	Wheeler et al., 1969
					Peeled roots	Endrin	0.02 (F)	
					Root peels	Endrin	0.03 (F)	
	2		0.74		Greens	Endrin	0.01 (F)	
					Peeled roots	Endrin	0.02 (F)	
					Root peels	Endrin	0.07 (F)	
	4		1.98		Greens	Endrin	0.02 (F)	
					Peeled roots	Endrin	0.04 (F)	
					Root peels	Endrin	0.12 (F)	
Potato	3			19 weeks	Tubers	Endrin	0.084–0.117 (F)	Winnett and Reed, 1968
	6			19 weeks	Tubers	Endrin	0.100–0.133 (F)	

[a] At time of planting.

[b] Based on fresh (F) or dry (D) weight.

[c] Time from endrin application to harvest.

[d] Endrin applied twice at 2 lbs/acre: 11 months and 6 months before harvest.

and cucumber (*Cucumis sativus*) in pots in a greenhouse. The soil contained 0.5 or 5.0 ppm endrin. The mean concentration of endrin in aerial parts of three-week-old seedlings varied considerably with the species, ranging from 0.045 ppm in soybean to 0.304 ppm in bromegrass for plants grown in soil that contained 0.5 ppm endrin. For all the plants except cucumber, however, endrin uptake by each species was proportional to the concentration of endrin in soil; that is, mean endrin residues in plants grown in soil containing 5.0 ppm endrin were tenfold the residues found when soil concentration was 0.5 ppm (Tables 4.1 and 4.2).

Harris and Sans (1967) noted that soil type as well as plant species affects the amount of endrin absorption. When carrot, radish, turnip, and onion (*Allium cepa*) were field grown in sandy loam soils containing 0.12 ppm endrin, no endrin residues were detected in the plants. With muck soils that contained 8.91 ppm endrin, endrin residues were detected in carrots (0.06 ppm) and in radishes (0.04 ppm), but not in turnips or onions. The extensive experiments of Beall and Nash (1969) are probably more indicative of the effect of soil type on root uptake of endrin because the plants were grown in pots under controlled greenhouse conditions (Table 4.2). Plants grown in sandy loam usually had the highest endrin residues, whereas silt generally retarded endrin uptake.

It should be noted that the reported levels of endrin residues in plants are probably of little value in a study of plant metabolism of endrin. Hill (1971) emphasized that the dissipation of residues from growing crops results from the combined effects of various factors, including pesticide volatilization, photodegradation, mechanical removal by wind and rain, metabolism, and dilution effected by plant growth. Hill concluded that it cannot be generalized that a given pesticide will always disappear from a given crop at the same rate.

Hill's conclusions are supported by the experiments of Korte (1967) and co-workers (Weisgerber et al., 1968). Although the same researchers used the same plant species in experiments in which ^{14}C-endrin was applied to the leaves of cabbage (*Brassica oleracea capitata*), the amount of radioactivity recovered after four weeks was 6% in one experiment and 25% in the other. The amount of total recovered radioactivity is, therefore, probably not significant.

The value of experiments of this type is, therefore, primarily in the quantitative data, that is, the presence or absence of endrin and/or its metabolites in the different plant parts, identification of the various compounds, the relative amounts of the compounds at the different locations, and the relative quantities of parent compounds and their metabolites and/or degradation products.

Foliar absorption of endrin and its subsequent translocation within the plant were demonstrated experimentally by a number of investigators. In these experiments, endrin was applied directly to the leaves; the endrin often contained ^{14}C as a radioactive label. Radioactivity was subsequently detected in unaltered endrin and in its metabolites as well. The data are summarized in Table 4.3.

Table 4.2. Plant uptake of endrin from soils

Soil	Soil type	Endrin residue (ppm)[a]				
		Soybean	Corn	Alfalfa	Bromegrass	Cucumber
Endrin concentration in soil, 0.5 ppm						
Lakeland	Sandy loam	0.066	0.084	0.424	0.249	0.206
Hagerstown	Sandy clay loam	0.030	0.067	0.211	0.259	0.242
Chillum	Silty loam	0.032	0.056	0.086	0.219	0.124
Sterling	Loam	0.038	0.062	0.179	0.333	0.228
Sharkey	Clay	0.060	0.069	0.174	0.462	0.238
	Mean	0.045	0.068	0.215	0.304	0.208
Endrin concentration in soil, 5.0 ppm						
Lakeland	Sandy loam	0.594	1.132	5.989	3.912	1.734
Hagerstown	Silty clay loam	0.260	0.461	1.037	1.659	1.118
Chillum	Silty loam	0.256	0.497	1.330	2.031	0.670
Sterling	Loam	0.365	0.575	1.407	3.020	0.812
Sharkey	Clay	0.575	0.875	2.556	5.014	*b*
	Mean	0.410	0.708	2.464	3.127	1.084

[a] Based on dry weight of three-week-old (soybean, corn, alfalfa, bromegrass) and four-week-old (cucumber) seedlings.

[b] Seedlings killed by disease.

Source: M. L. Beall, Jr. and R. G. Nash, *Argon. J.* 61(4): 571-575 (1969. Copyright 1970 American Society of Agronomy.

Table 4.3. Distribution of endrin and its metabolites in plants following foliar application of ^{14}C-endrin

Plant	Endrin applied	Growth period (weeks)	Recovered radioactivity (%)	Location	Compound	Reference
Cotton	120 ppm	12	33	On and in leaves, 80%; in stalks, pods, fibers, soil, 20%; in roots and seeds, traces	Endrin + 4 metabolites; main metabolite: slightly more hydrophilic than endrin; 3 metabolites: highly polar	Hill, 1970
Cotton	200 μg/leaf			In leaves	Ketoendrin; endrin aldehyde, traces	Soto and Deichmann, 1967
Cotton	12.6 mg/leaf	12	33	On and in leaves, 79.7%; on stalks, pods, soil, 20.3%; in fibers and seeds, traces	Endrin + 5 metabolites; 3 metabolites: slightly more hydrophilic than endrin (1 = ketoendrin); 2 metabolites: very hydrophilic	Bayless et al., 1970
Cotton					Endrin + metabolites; some metabolites: slightly more hydrophilic than endrin; other metabolites: very hydrophilic	Hill, 1971
Cabbage	60 ppm	4	6	On and in leaves, 85.3%; in soil, 9.2%; on and in stems and roots, 5.5%	Endrin + 2 metabolites; main metabolite: very hydrophilic, 95%; ketoendrin, 5%	Korte, 1967; Weisgerber et al., 1968
	5 ppm	2	33.6	On plant surfaces, 1.2%; inside plants, 95.2%; in soil, 3.6%		
Cabbage	5 ppm	3	30.1	On plant surfaces, 1.2%; inside plants, 93.8%; in soil, 5.0%		Korte, 1967; Weisgerber et al., 1968
		4	25.0	On plant surfaces, 1.0%; inside plants, 92.5%; in soil, 6.4%		
Cabbage	1.3 mg/plant (60 ppm)	4	6	On leaf surfaces, 13.6%; inside leaves, 74.6%; in stems and roots, 3.4%; in soil, 8.5%	Endrin, 90%; 2 metabolites, 10%; main: very hydrophilic; minor: ketoendrin	Korte et al., 1970
	75 ppm	4	30		Endrin, 91%; 2 metabolites, 9%; main: very hydrophilic; minor: ketoendrin	
Cabbage	500 μg/plant			Leaves, stems, roots, soil	Endrin, 90%; metabolites, 10%	Brooks, 1969

Table 4.3 (continued)

Plant	Endrin applied	Growth period (weeks)	Recovered radioactivity (%)	Location	Compound	Reference
Cabbage	1.3 mg/plant	4	11		Endrin, 90%; a very polar metabolite, 10%; Δ-ketoendrin, small amt	Brooks, 1974b
Tobacco	52 ppm	4	36	On leaf surfaces, 80.6%; in plants, 19.4%	Endrin, 97.7%; 2 metabolites, 2.3%; main: very hydrophilic; minor: ketoendrin	Korte et al., 1970
Tobacco	2.08 mg/plant (100 ppm)	1	73–79	On and in plants	Endrin, extremely hydrophilic metabolite(s)	Weisgerber et al., 1969
		2	61–66			
		4	42–47			
		6	32–47			
	1.04 mg/plant (50 ppm)	1	75	On and in plants	Endrin, extremely hydrophilic metabolite(s)	
		2	70			
		4	36			
		6	34			
Carrot	14 μg/plant	3			Endrin, 68%; 2 metabolites, 32%; main: very hydrophilic; minor: ketoendrin	Korte et al., 1970

Hill (1970) reported that when about 120 ppm ^{14}C-endrin was applied to leaves of cotton (*Gossypium hirsutum*) plants in three applications, 33% of the applied radioactivity was recovered after 12 weeks. Of the recovered radioactivity, 80% was found on and in the leaves; 20% was in the stalks, pods, and fibers and in the soil; only traces were detected in the roots and seeds. The radioactivity compounds consisted of endrin and four metabolites. The main metabolite was only slightly more hydrophilic than endrin; the other three were highly polar. None of the metabolites was identified.

The work of Burton and Potter demonstrated that endrin is rapidly absorbed by leaves of cotton (Soto and Deichmann, 1967). Two hours after 200 µg endrin was brushed onto each leaf of a cotton plant, ketoendrin was detected in the leaves at a concentration of 0.35 ppm; after seven days the concentration had decreased to less than 0.008 ppm. Traces of endrin aldehyde were also detected in the leaves. Since the endrin used in the experiment contained about 2% ketoendrin as well as traces of other impurities, the investigators felt that the endrin aldehyde was not necessarily a plant metabolite of endrin, that it may have been a decomposition product or a metabolite of one of the impurities.

In their experiments, Bayless et al. (1970) applied 12.6 mg ^{14}C-endrin in three equal portions to the upper surfaces of cotton plant leaves. Twelve weeks after the last application, 33% of the applied radioactivity was recovered, primarily on and in the leaves and in the soil (Table 4.4). Of the recovered radioactivity, 76% was associated with endrin, 24% with endrin metabolites. The five metabolites were of two types; those in group 1 were slightly more hydrophilic than endrin. One of the three components was identified as ketoendrin; the two components of group 2 were very hydrophilic.

Hill (1971) reported that when endrin was applied to the leaves of cotton, endrin as well as degradation products was subsequently detected in the plants. There were two groups of endrin metabolites — one slightly more hydrophilic than endrin, the other very hydrophilic.

When deuterated endrin was applied to cotton, penetration of endrin into the plants was not detected (Korte et al., 1970). Small amounts of ketoendrin were found on leaf surfaces; this compound was attributed to ultraviolet rearrangement of endrin.

Working with young cabbage plants which had not yet formed heads, Korte (1967) and co-workers (Weisgerber et al., 1968) demonstrated that endrin is absorbed by the leaves and then translocated throughout the plant and even into the soil. When 60 ppm ^{14}C-endrin in acetone was applied to the cabbage leaves, 6% of the applied radioactivity was recovered four weeks later. Almost all of the recovered radioactivity was found within the plant or in the soil, and there were only traces on the plant surfaces. There was a gradient in the recovered radioactivity — the concentration was greatest in the leaves; it diminished through the stems and then the roots and was least in the soil (Table 4.5). There

Table 4.4. Distribution and transformation of endrin 12 weeks after application of 12.6 mg ^{14}C-endrin to leaves of cotton (*Gossypium hirsutum*)

Location	Concentration (ppm)	Radioactivity[a] (%)	Endrin (%)	Metabolites (%)		
				Group 1	Group 2	Total
Dead leaves						
Surface	129.0	31.5	75	19	6	25
Interior	72.2	17.7	75	12	13	25
Living leaves						
Surface	19.5	16.2	79	15	6	21
Interior	17.1	14.3	71	20	9	29
Stalks	0.33	0.27	85	12	3	15
Roots	ND[b]	ND				
Pods	4.9	0.06	40[c]		10	>10
Fibers	0.36	0.003	88[c]		12	>12
Seeds	0.033	0.0006				
Soil	0.0053	20.0	81	16	3	19

[a] As percentage of total recovered radioactivity.

[b] ND, not detected.

[c] Endrin and group 1 metabolites could not be distinguished.

Source: Reprinted with permission from *Tetrahedron* 26(3): 775-778 (1970). A Bayless, I. Weisgerber, W. Klein, and F. Korte, "Contributions to Ecological Chemistry XXV. Conversion and Residue Behavior of 14-C-Endrin in Cotton." Copyright 1970, Pergamon Press, Ltd.

Table 4.5. Concentration, distribution, and transformation of endrin as measured four weeks after application of 60 ppm ^{14}C-endrin to leaves of cabbage (*Brassica oleracea capitata*)

Location	Concentration (ppm)	Radioactivity[a] (%)	Metabolites[b] (%)
Withered leaves			
Surface	2.55	3.9	∿1
Interior	5.10	8.1	∿1
Living leaves			
Surface	0.26	9.4	∿1
Interior	1.80	63.9	7
Stem			
Surface	0.13	1.5	∿1
Interior	0.26	2.8	26
Root			
Surface	0.004	0.3	∿2
Interior	0.09	0.9	40
Soil	0.06	9.2	51

[a] As percentage of total recovered radioactivity.

[b] As percentage of radioactive compounds in each plant part.

Source: I. Weisgerber, W. Klein, A. Djirsarai, and F. Korte, *Justus Liebigs Ann. Chem.* 713: 175-179 (1968). Copyright 1968 Verlag Chemie GmbH.

was also a gradient in the ratio of metabolite concentration to endrin concentration; this gradient, however, was in the reverse order. Whereas only 7% of the recovered radioactivity in leaves was associated with endrin metabolites, metabolites accounted for 26% of the radioactivity in stems, 40% in roots, and 51% in the soil. Of the total recovered radioactivity, 10.2% was attributable to the metabolites. Two metabolites were distinguished; the main metabolite (95%) was very hydrophilic. The other metabolite (5%) was slightly more hydrophilic than endrin; on the basis of its chromatographic behavior, it was identified as ketoendrin.

The investigators demonstrated that these radioactive compounds were products of plant metabolism of endrin. In plants kept in the dark for four weeks after the application of endrin, the concentrations of metabolites were about the same as in plants kept in the light. Consequently, the compounds could not have been the products of photodecomposition of endrin. Korte and co-workers also determined that decomposition on active surfaces in the presence of light and air could not account for the metabolities — less than 1% of the endrin applied to leaf surfaces, faded leaves, glass plates, and silica gel plates degraded in four weeks under the conditions of the experiment. Furthermore, the metabolites detected in the soil had been excreted by the plants, since, under the experimental conditions, less than 4% of endrin in soil degrades in four weeks.

In another experiment with cabbage, Weisgerber et al. (1968) followed the effect of time on the recovery of radioactivity. Carbon-14-endrin was applied to the leaves at the rate of 5 ppm based on total plant weight. Recovery of radioactivity decreased from 33.6% after two weeks to 25.0% after four weeks (Table 4.6). During the same time interval, the amounts of recovered radioactivity decreased on plant surfaces (from 0.40 to 0.26%) and inside the plants (from 32.0 to 23.1%), while it increased in the soil (from 1.2 to 1.6%).

Table 4.6. Distribution of radioactivity after application of 5 ppm ^{14}C-endrin to leaves of cabbage (*Brassica oleracea capitata*)

Time after application (weeks)	Recovered radioactivity (%)			
	On plant surface	Inside plants	In soil	Total
2	0.40	32.0	1.2	33.6
3	0.35	28.25	1.5	30.1
4	0.26	23.10	1.6	25.0

Source: I. Weisberger, W. Klein, A. Djirsarai, and F. Korte, *Justus Liebigs Ann. Chem.* 713: 175-179 (1968). Copyright 1968 Verlag Chemie GmbH.

Subsequent experiments with cabbage revealed that 1 hr after application to upper leaf surfaces of 1.3 mg ^{14}C-endrin in acetone per plant (which corresponds to 60 ppm based on plant weight at end of experiment), 100% of the radioactivity was still present on the leaf surfaces (Korte et al., 1970). After the plants had grown four weeks in greenhouses, about 6% of the applied radioactivity was recovered: 0.8% on leaf surfaces, 4.4% inside the leaves, 0.2% in the stems and roots, and 0.5% in the soil. On the basis of loss of radioactivity from surfaces (glass, silica gel, paper), the disappearance of 60% of the applied radioactivity was attributed to vaporization of endrin from the leaf surfaces. The remainder of lost radioactivity was attributed to removal from the plants by processes such as transpiration and excretion with guttation water. The metabolic rate (i.e., the proportion of radioactivity recovered in plants and soil that is attributable to metabolites) was 10%. When the cabbage plants were kept in the dark after application of endrin, the metabolic rate was 14%. Conversion on active surfaces (dead leaves, glass plates, silica gel, and plant homogenates) amounted to less than 2%, so the conversion products detected in and on the plants were endrin metabolites. Two metabolites were detected — the main one was very hydrophilic, the other was ketoendrin.

When 75 ppm ^{14}C-endrin was applied to the upper surfaces of cabbage leaves, 30% of the applied radioactivity was detected four weeks later (Korte et al., 1970). Nine percent of the recovered radioactivity was associated with hydrophilic metabolites of endrin.

Brooks (1969) noted several of Korte's experiments with cabbage. In one experiment, when 50 μg ^{14}C-endrin was applied to leaves of growing cabbage plants, 66% of the applied radioactivity was lost by evaporation in two weeks. In another experiment, after 500 μg ^{14}C-endrin was applied per plant, radioactivity was recovered, in decreasing order, from the leaves, stems, roots and soil. The radioactive material recovered on plant surfaces and in the leaves was mostly endrin. The proportion of endrin metabolites, which constituted 10% of the recovered activity, increased progressively in stems, roots, and soil. Brooks suggested two explanations for this finding: (1) water-insoluble endrin is retained in the leaves, while the hydrophilic conversion products are transported to other parts of the plant; (2) metabolism of endrin occurs primarily in the stems and roots.

Brooks (1974b) reported that when 1.3 mg ^{14}C-endrin per plant was applied to the leaves of cabbage, 11% of the applied radioactivity was recovered after four weeks. Of the recovered radioactivity, 90% was associated with endrin, 10% with a very polar metabolite, and a small amount with delta-ketoendrin.

Most of the experimental work on the tobacco was done by Korte and co-workers. When 52 ppm ^{14}C-endrin was applied to tobacco leaves, 36% of the applied radioactivity was recovered after four weeks: 29% on leaf surfaces and 7% in the plants; the metabolic rate was 2.3%. The metabolite fraction consisted of two compounds — a very hydrophilic main metabolite and some ketoendrin (Korte et al., 1970). When ^{14}C-endrin was applied to the upper surfaces of tobacco leaves at the rates of 2.08 and 1.04 mg per plant (100 and 50 ppm respectively), 34 and 47% of the applied radioactivity was recovered on and in the treated plants after six weeks (Table 4.7). The investigators concluded that part of the applied endrin was eliminated by the plants unaltered into the atmosphere, while the roots excreted both unaltered endrin and endrin metabolite(s) into the soil. The radioactive compounds consisted of endrin and at least one extremely hydrophilic metabolite, which was chromatographically identical to the main metabolite found in cabbage; it was neither ketoendrin nor endrin aldehyde (Weisgerber et al., 1969).

In their work with carrot, Korte et al. (1970) found that the metabolic rate depended on the mode of application. In these experiments, 14 μg ^{14}C-endrin was applied per plant, and the material was analyzed three weeks later. When the endrin was applied to the soil, the metabolic rate was 5%. On the other hand, the metabolic rate was 12% when endrin was injected into the roots and 32% when it was applied to the leaves near the vegetation point. Two metabolites of endrin were distinguished — a main metabolite that was very hydrophilic and also some ketoendrin.

Some experiments were designed specifically to demonstrate, and also to compare, uptake of soil endrin by roots and of vaporized endrin by leaves (Nash and Beall, 1970; Beall and Nash, 1971). Soybean plants were grown in pots in a greenhouse for 53 days. Either the surface or the subsurface soil layer was treated with endrin (concentration: 20 ppm),

Table 4.7. Distribution and metabolism of ^{14}C-endrin after application to upper surfaces of leaves of tobacco (*Nicotiana tabacum*)

Growth period	Recovered radioactivity (%)			Metabolic rate (%)
	Total	Surface^a	Internal^a	
	Endrin application: 2.08 mg/plant (100 ppm)			
2 days	81.2	93.8	6.2	
1 week	97.2	93.8	6.2	4.6
2 weeks	66.3	92.2	7.8	9.8
4 weeks	46.7	71.2	28.8	6.6
6 weeks	47.0	73.5	26.5	8.9
	Endrin application: 1.04 mg/plant (50 ppm)			
2 days	87.9	96.3	3.7	
1 week	75.2	95.2	4.8	3.9
2 weeks	69.6	90.1	9.9	5.8
4 weeks	36.4	80.7	19.3	20.8
6 weeks	34.3	42.4	57.6	7.2

^{a}Based on radioactivity recovered on and in plants.

Source: I. Weisgerber, W. Klein, and F. Korte, *Liebigs Ann. Chem.* 729: 193-197 (1969). Copyright 1969 Verlag Chemie GmbH.

and the two were separated by a water- and vapor-impermeable barrier that prevented movement of endrin through the soil from one layer to the other. A glass tube that extended upward from the barrier isolated the stem of each plant from the surrounding surface soil layer. Each pot was enclosed in a polyethylene cage in order to localize the vapors around the plant. These experiments demonstrated that soybean roots take up endrin from soil and that leaves absorb endrin that has vaporized from treated soil (Table 4.8). With root sorption, endrin concentrations were highest in the stems and then, in decreasing order, in leaves, pods, and seeds. When endrin vapor was sorbed, the concentrations were highest in the leaves, then decreased through the pods, stems, and seeds. Endrin as well as two metabolites was detected in the plants. The ratio of metabolites to parent endrin was greater in leaves than in stems.

When seedlings of wheat were grown for 16 days in water that contained 0.11 ppm ^{14}C-endrin, radioactivity was recovered in both water and seedlings (Klein et al., 1968b). Endrin metabolites accounted for 58% of the radioactivity in the water and 20% of that in the plants.

The endrin applied to crops and soils is seldom chemically pure, and related compounds such as ketoendrin are frequently present as contaminants and/or degradation products. Klein (1972) investigated the fate of delta-ketoendrin applied to leaves of cabbage. He found that ketoendrin residues disappeared more slowly than did those of endrin. The metabolic

Table 4.8. Sorption of endrin by soybean (*Glycine max*) from soil and from air

	Endrin concentration (ppm)	
Plant part	Root sorption[a]	Leaf sorption[b]
Upper leaves	87.71	15.95
Lower leaves	160.55	33.78
Upper stems	217.30	2.42
Lower stems	359.62	1.66
Pods	10.44	2.84
Seeds	3.30	0.99
Average[c]	38.1	6.7

[a] Sorption of endrin from soil.

[b] Sorption of endrin vaporized from soil.

[c] Average concentration in aerial parts of plants.

Source: M. L. Beall and R. G. Nash, *Agron. J.* 63(3): 460-464 (1971). Copyright 1971 American Society of Agronomy.

rate in cabbage, four weeks after the application, was greater for ketoendrin (15%) than for endrin (10%). The main metabolite of ketoendrin, an unidentified polar compound, was present in highest concentrations in leaves and stems.

In 1967, Nash and Harris (1973) examined the soil in some fields that had been treated in 1951 with endrin at the rates of about 56 and 224 lb/acre (56 and 225 kg/ha; soil concentration, about 28 and 112 ppm). Endrin, ketoendrin, two endrin aldehydes, and endrin alcohol were identified in the soil. When soybean crops were grown in these fields, endrin, ketoendrin, one endrin aldehyde, and endrin alcohol residues were detected in the beans. Whereas the concentrations of endrin and ketoendrin in soybeans were about 0.2 and 0.5% of the respective concentrations in the soil, the endrin alcohol concentration in soybeans was about 10% of its concentration in soil. The reason for the relatively greater concentration of endrin alcohol in soybeans has not been ascertained. Endrin alcohol may be more readily taken up from the soil and translocated within the plant. Another possibility is that endrin, ketoendrin, and possibly endrin aldehyde are metabolized by plants to endrin alcohol (it is known that endrin and ketoendrin are converted to the alcohol under severe conditions of acid and heat).

It has been demonstrated that plants take up endrin from the soil by root absorption and that leaves take up endrin applied directly to leaf surfaces as well as vaporized endrin. Following uptake, endrin is translocated to all parts of the plant. Endrin taken up by specific root sections is subsequently translocated both acropetally and basipetally (Beall and Nash, 1972). Plants metabolize endrin to delta-ketoendrin and possibly to endrin aldehyde and as three additional metabolites (Brooks, 1974b). The reported metabolites are of two types — one group, including ketoendrin, is slightly more hydrophilic than endrin, whereas the other is very hydrophilic.

4.3.2 Effects of Endrin

The major use of endrin is as a pesticide in agriculture (see Sect. 2.5). The compound is usually applied to the fields by spraying or dusting, and in this way it comes in direct contact with the plants. It is essential that the plants suffer no adverse effects from this treatment, and the paucity of information in the literature about the effects of endrin on plants is therefore surprising.

Scholes (1955), in the 1951 progress reports by Julius Hyman and Company on the development of endrin, noted that slight burning of corn buds and cucumber foliage occured when plants were sprayed with endrin.

In his review of cholorinated insecticides, Brooks (1974a) reported that "phytotoxicity appears to present few problems" if endrin is properly applied. Adverse effects noted with some sensitive plants were traced to the solvent used in the formulation. Endrin itself can be injurious to corn (*Zea mays*).

Cucurbits (plants in the gourd family) are highly sensitive to chemicals and were therefore selected by Sood et al. (1972) for use in a study of the phytotoxicity of endrin. Bottlegourd (*Lagenaria siceraria*), cucumber (*Cucumis sativus*), and tinda (*Citrullus vulgaris*) plants at the four-leaf stage were sprayed once with a 0.03 or a 0.04% endrin emulsion. Phytotoxicity was assessed by the number of spots on the foliage that resulted from scorching. The 0.03% endrin emulsion was slightly toxic to cucumber, but highly toxic to bottlegourd and tinda by the seventh day. With the 0.04% endrin emulsion, high phytotoxicity to all three varieties was apparent by the third day after spraying.

Abo El Liel et al. (1970) studied the effects of various insecticides on Ashmouni cotton (*Gossypium hirsutum*). They concluded that, when endrin in a mixture with DDT-lindane is sprayed on cotton plants four to ten times during the growing season, there are no undesirable effects which could reduce the yield of cotton seed. The application consisted of 1.5 liters of endrin plus 1.5 liters of DDT-lindane per feddan (1 feddan is about 1 acre or 0.4 ha); insecticide concentrations were not reported.

Crouch and Radwan (1972) investigated the use of endrin in a coating for Douglas fir (*Pseudotsuga menziesii*) seed as protection against seed-eating deer mice. At 0.5%, endrin had little effect on seed germination. Of the endrin-coated seeds, 82% germinated on perlite and 73% in soil; for control (i.e., untreated) seeds, the germination rates were 83 and 80% respectively. Seedling growth was not affected by endrin.

Scholes (1955) investigated the effects of endrin on mitosis in onion (*Allium cepa*) roots. When onion seeds were germinated in petri dishes which contained endrin dust on gypsum at amounts equivalent to 0.4 and 2.0 lb/acre (about 0.4 and 2.0 kg/ha; soil concentration, about 0.2 and 1.0 ppm), there was very little effect on resting and on dividing cells examined after a 48-hr growth period. A slight stickiness of the chromosomes in anaphase delayed, but did not prevent, separation of the chromatids.

In another experiment (Scholes, 1955), endrin was applied to and mixed with the surface layer of soil at the heavy dose of 2.0 lb/acre (about 2.0 kg/ha; soil concentration, about 1.0 ppm). The onion (*Allium cepa*) seedlings were examined ten days after sowing. Rates of germination and growth were within the normal variation for untreated seed. Cytological examination of root cells did not provide any evidence of toxic effects on the resting cell. In mitotic cells, there was an increased number of anaphases with incomplete separation of chromatids, but this was not sufficient to affect the growth rate.

Wuu and Grant (1966) investigated the phytotoxicity of endrin to barley (*Hordeum vulgare*). Barley seeds were soaked in endrin solutions at 500, 1000, or 1500 ppm for 6, 12, or 24 hr. They were then washed in tap water, placed in petri dishes, and incubated. Germinability of the seeds, as measured after 36 hr, decreased with increase in endrin concentration and lengthening of the soaking period. Duration of soaking had the greater effect. The germination rate was severely reduced in seeds soaked in endrin for 24 hr; at endrin concentrations of 500 and 1500 ppm, only 52 and 50% respectively, of treated seeds germinated (see Table 4.9 for a tabulation of the data). Cytological studies of root-tip cells confirmed the germination data. The mean frequency of cells with chromosome aberrations was 6.01, 7.63, and 9.05% for the 6-hr, 12-hr, and 24-hr treatment periods respectively. The overall mean was 7.59% for endrin-treated barley seeds. The most common chromosome aberrations were fragments in metaphase and in anaphase, and anaphase bridges. There were no obvious morphological effects on root growth with any treatment.

In order to study the transmission of chromosomal abnormalities, Wuu and Grant (1966) soaked barley (*Hordeum vulgare*) seeds in a 1000-ppm solution of endrin for 12 hr; the seeds were then washed in tap water and planted in soil. Seeds of the C-1 generation were harvested and planted. Among the 933 seedlings in the C-2 generation, there were three mutants (one plant with a striped leaf and two dwarf plants). The mutation rate

Table 4.9. Effect on barley seeds (*Hordeum volgare*) of soaking in endrin solutions

Soaking period	6 hr			12 hr			24 hr		
Concentration (ppm)	500	1000	1500	500	1000	1500	500	1000	1500
Germination (%)	92	88	90	84	84	80	52	80	50
Abnormal cells (%)	5.58	6.42	6.02	7.48	8.54	6.53	8.96	7.54	10.63
Chromosome aberrations (No. of cells)									
Metaphase fragments									
Single	4	2	6	8	11	6	9	7	4
Double	0	1	1	3	3	0	1	0	1
Multiple	0	0	2	1	2	3	1	1	4
Anaphase bridges									
Single	3	4	4	3	5	2	4	3	8
Anaphase fragments									
Single	4	1	0	1	0	1	3	3	6
Double	0	1	0	1	1	1	0	3	1
Multiple	1	0	1	0	0	0	0	0	0
Other[a]	0	5	1	2	2	0	1	2	3
Total	12	14	15	19	24	13	19	19	27

[a] Includes telophase bridges, lagging chromosomes, bridges and fragments, and incomplete chromosome breakages.

Source: K. D. Wuu and W. F. Grant, *Can. J. Gent. Cytol.* 8(3): 481-501 (1966). Copyright 1966 Genetics Society of Canada.

was therefore 0.32%, whereas it was 0.11% in the control group (1 yellow plant out of 891). Only 1.55% of the root-tip cells examined had chromosome aberrations, whereas the frequency in the C-1 generation had been 8.54%.

Wuu and Grant speculated that possible nonviability of cells with chromosome aberrations resulted in the low transmission of chromosomal abnormalities from the C-1 to the C-2 generation. Cells with aberrations of a lethal nature would have been eliminated in the course of the numerous cell divisions which had occurred, and only chromosome aberrations with less severe effects would have persisted.

Wuu and Grant (1967b) also investigated endrin induction of chromosome aberrations in root-tip cells of broad bean (*Vicia faba*). Seedlings that were 15 cm high (10 to 15 days old) were carefully removed from the soil and immersed in aqueous endrin solutions for 3, 6, or 12 hr. After washing and 24 hr in tap water, the roots were fixed and stained. Endrin at concentrations of 200, 400, and 600 ppm was extremely toxic and strongly inhibited root growth. Even at concentrations of 100, 200, and 300 ppm, endrin greatly increased the frequency of chromosome aberrations. The number of chromosomal abnormalities increased with increase in endrin concentration and also with increase in duration of treatment. The mean frequency of endrin-induced abnormal cells was 18.55%.

The effect of endrin on meiotic cells of barley (*Hordeum vulgare*) was also studied by Wuu and Grant (1967a). Barley seeds were soaked in a 1000-ppm solution of endrin for 12 hr; they were then washed in tap water and sown in soil. Seeds of the C-1 generation were harvested and sown. Pollen mother cells of the C-1 and C-2 generations were examined, and the frequency of cells with chromosome aberrations was 0.26 and 0.37% respectively. These frequencies were not significantly different from those in control plants. In another experiment, barley seeds were soaked 12 hr in tap water and sown in soil; then on day 16 the seedlings were sprayed with a 500-ppm solution of endrin at the rate of 2 ml per five seedlings. Chromosome aberrations (anaphase bridges and fragments) appeared in the pollen mother cells at a frequency of 0.98%. Spraying barley plants with endrin at 500 ppm, therefore, induced a higher frequency of chromosomal abnormalities in meiotic cells than did soaking the seeds in endrin at 1000 ppm for 12 hr.

In addition to these studies of the phytotoxicity of endrin, there have also been some studies of the effects of endrin-contaminated soil on various constituents of plants growing in such soil.

Thakre and Saxena (1970) reported that the presence of endrin in the soil at 10, 20, and 30 ppm affected calcium uptake by plants. Endrin at these concentrations reduced calcium uptake by wheat but more than doubled the calcium uptake by corn. In the same experiments, 10 ppm endrin did not affect iron uptake, whereas 20 and 30 ppm did reduce iron uptake significantly in wheat and slightly in corn. Wheat plants were harvested after 90 days of growth, corn after 50 days.

Endrin in the soil at concentrations of 10, 20, and 30 ppm also affected the amino acid composition of corn harvested after 50 days of growth (Thakre and Saxena, 1972). The most significant changes were increases in the contents of proline, lysine, arginine plus histidine, and leucine, and decreases in the contents of tryptophan and valine. Thakre and Saxena speculated that the presence of endrin in soil and its absorption by plants could alter the nutritional value of these plants as a consequence of changes in the amino acid composition.

Cole et al. (1968) investigated the effects of different concentrations of endrin in soil on the growth and on the macro- and microelement contents of corn and green beans (*Phaseolus vulgaris*) (see Table 4.10). The aboveground portions of the plants were analyzed after four and eight weeks of growth. Endrin at all concentrations significantly reduced the phosphorus contents of eight-week-old corn plants and of four- and eight-week-old bean plants. The potassium content of all four-week-old bean plants was decreased, but this change was noted in eight-week-old bean plants only when the endrin concentration was 1 ppm. Endrin increased the magnesium content of corn but decreased it in bean. With the microelements, endrin effected significant decreases in the contents of copper and zinc in both corn and bean and of boron in bean. Significant increases in the levels of manganese and iron were detected. The effect of endrin on the fresh weight was only slight in four-week-old plants; however, the decreases were significant in eight-week-old corn plants grown in soil that contained 100 ppm endrin and in eight-week-old bean plants grown in soil with 10 and 100 ppm endrin.

Table 4.10. Effect of soil endrin on aboveground portions of corn (*Zea mays*) and bean (*Vicia faba*)

Plant	Endrin (ppm)	Fresh weight (g)	Macroelements (% of dry weight)					Microelements (ppm of dry weight)						
			N	P	K	Ca	Mg	Mn	Fe	Cu	B	Al	Sr	Zn
Corn, 4 weeks	0	4.1	6.02	0.88	6.80	0.80	0.29	434	185	13.4	19	121	11	117
	1	3.8	5.68	0.71[a]	5.84	0.87	0.31	454[a]	205	11.1[a]	25[a]	166	11	83[a]
	10	4.0	5.67	0.88	6.30[a]	0.89	0.33[a]	533[a]	242[a]	12.8	21	137	11	91[a]
	100	4.2	5.64	0.84	6.08	0.94	0.32	460	171	10.9[a]	18	117	12	96[a]
Corn, 8 weeks	0	27.1	3.45	0.54	4.71	0.73	0.27	362	119	8.5	15	74	9	65
	1	27.8	3.43	0.44[a]	5.00	0.75	0.25	331	117	8.5	16	63	10	40[a]
	10	24.8	3.20	0.48[a]	5.53	0.68	0.26	350	120	7.9	16	78	10	49
	100	23.1[a]	3.15	0.46[a]	4.87	0.84	0.30[a]	318	124	7.6[a]	15	78	11[a]	51
Bean, 4 weeks	0	7.0	6.25	0.65	5.40	1.82	0.40	513	257	10.6	41	205	23	96
	1	6.0	6.25	0.45[a]	5.00[a]	1.73	0.37[a]	499	224	9.1[a]	33[a]	171	22	75[a]
	10	6.4	6.45	0.50[a]	4.95[a]	1.83	0.40	592[a]	275	10.0	35[a]	210	22	94
	100	6.0	6.09	0.60[a]	4.89[a]	1.94	0.38	574	259	11.2	39	186	22	79[a]
Bean, 8 weeks	0	18.8	3.92	0.54	3.82	1.74	0.36	523	233	10.5	59	132	22	76
	1	18.0	3.28[a]	0.36[a]	2.95[a]	1.98	0.36	495	244	7.7[a]	38[a]	177	21	51[a]
	10	12.8[a]	3.83	0.38[a]	3.90	1.52	0.32[a]	495	305[a]	8.2[a]	38[a]	198	20[a]	62
	100	12.4[a]	4.24	0.47[a]	3.93	1.88	0.37	558	234	8.2[a]	43[a]	119	22	69

[a] Significantly different from the control.

Source: modified from Cole et al., 1968.

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5. BIOLOGICAL ASPECTS IN ANIMALS

5.1 SUMMARY

Animals are exposed to endrin via all media (i.e., food, water, air, soil). Endrin is absorbed through the digestive tract and through body surfaces. Absorption of endrin is based on solubility differences between water and liquid, with a continual residue exchange between tissues and body fluids or, in the case of aquatic animals, the surrounding medium. After an initial rapid uptake, endrin accumulation reaches a plateau that appears to represent a balance between elimination and uptake. Both rate of uptake and equilibrium concentrations are directly related to the exposure level.

Endrin is not stored in tissues, but remains in a dynamic state, equilibrating with ambient levels. After an equilibrium concentration is reached, duration of exposure does not affect the amount of residue. However, after chronic exposures, many insects, shrimp, and fish develop a tolerance to increased endrin doses.

Evidence for transformation of endrin by aquatic animals and birds is derived from "balance" studies that show incomplete recovery of the introduced endrin and the presence of polar metabolites. Identification of the metabolites in fish and aquatic invertebrates is speculative, but a hydroxylated compound has been suggested by analogy with dieldrin metabolism.

Endrin is eliminated from birds, mammals, and aquatic animals when the exposure level is lowered. Passive equilibrium reactions that lower residues in tissues appear to account for the major pathway of endrin elimination; however, active excretion has been suggested to occur in bluegill sunfish. The rate of endrin accumulation in bluegills drops after initial uptake, possibly resulting from stimulation of an elimination mechanism.

Aquatic invertebrates and fish are killed by endrin concentrations of 1 ppb in water, while birds and mammals, acquiring endrin from food, show mortality at doses from 2 to 20 ppm. Concentrations at a lower magnitude caused pathologic and reproductive changes, suggesting that chronic exposure to even lower concentrations may affect nerve function and metabolism.

The central nervous system appears to be the primary site of endrin toxicity in all animals. The symptoms of endrin poisoning reflect nerve stimulation, and reactions to acute levels generally follow the order: hypersensitivity, hyperactivity, tremors, loss of equilibrium, convulsions, accelerated breathing, and paralysis. Ganglia in insects and the telencephalic region of the brain in birds were identified as specific areas of endrin action. Brain cell membranes from endrin-resistant fish were found to have structural differences associated with increased endrin binding. Tissues of endrin-resistant insects also had higher endrin binding. Binding may decrease toxicity by lowering the amount of endrin entering the cells.

Other endrin effects include inhibition of certain enzymes and impairment of liver function. Increased serum osmolality, due in large part to high sodium and glucose levels, reflects the liver dysfunction. Stress factors, such as starvation, nonoptimal temperature, and disease, consistently tend to increase endrin toxicity. Low chronic exposure levels occasionally have the reverse effect of acute doses; for example, fecundity in red cotton bugs and growth of goldfish were increased at endrin concentrations below those causing inhibitory effects. Chronic endrin exposures reduced reproduction in fish and birds by decreasing egg production. Decreased hatchability and viability of bird eggs were also observed in some chronic feeding experiments (1 ppm endrin in the diet).

No evidence of tumors or cancer was found in dogs after chronic exposure to endrin (5 ppm in the daily diet). And no carcinogenicity or teratogenicity was reported elsewhere, but the paucity of research data is those areas makes any judgement inconclusive. Mutagenicity is suggested in fish by the inheritable tolerance to endrin, but mutations were ruled out in endrin-resistant insects.

5.2 AQUATIC ANIMALS

5.2.1 Invertebrates

5.2.1.1 Metabolism

5.2.1.1.1 Uptake and absorption. Aquatic mollusks and arthropods are exposured to endrin in the water, sediment, and food sources. Invertebrates act as extractors, continually exchanging residues between water and body fluids until an equilibrium is eventually reached (Hamelink, 1971). The equilibrium concentration in an organism will depend on the concentration of endrin in the water, species (physiology), bottom composition, and exposure time (Table 5.1).

Endrin is rapidly accumulated initially by mollusks and arthropods, achieving bioconcentration factors up to 49,000 and 2600 respectively (Metcalf et al., 1973). However, endrin levels decreased progressively after the initial uptake until equilibrium values were reached. This supports Hamelink's (1971) postulation that invertebrates exchange residues by physiological and chemical reactions until an equilibrium is attained in about five days. Direct absorption has also been suggested as contributing to body burdens of endrin (Metcalf et al., 1973).

Table 5.1. Endrin accumulation factors in aquatic invertebrates

Species	Water conc.	Exposure time (days)	Accumulation factor	Reference
Clam *(Mya arenaria)*	0.5 ppg	5	1,240	1
Clam *(Mercenaria mercenaria)*	0.5 ppb	5	480	1
	1.0 ppb	5	480	2
Mussel *(Hyridella australis)*	0.01 ppm	24	38	3
	0.5 ppm	3	7	3
Oyster (species not given)	0.001 ppm	10	1,000	4
Eastern oyster *(Crassostrea virginica)*	0.1 ppb	7	1,670	5
	0.05 ppm	7	2,780	5
Snail *(Physa sp.)*	0.01 ppm (plus contam. food)	33	49,218	6
Water flea *(Daphnia magna)*	1.0 ppb	1	2,600	6
		3	330	
Mosquito larva *(Culex pipiens quinquefasciatus)*	1.0 ppb	1	2,100	6
		3	310	

1. Butler, 1971.
2. Duke and Dumas, 1974.
3. Ryan et al., 1972.
4. Pimentel, 1971.
5. Mason and Rowe, 1976.
6. Metcalf et al., 1973.

Whole-body accumulations depend on ambient concentrations and exposure times. Endrin levels as much as 492, 3.44, and 2.6 ppm have been reported in soft tissue of snails, mussels, and clams respectively (Metcalf et al., 1973; Ryan et al., 1972; Butler, 1971).

5.2.1.1.2 Transformation and elimination. Biodegradability of endrin was determined using radiocarbon-labeled endrin in a model ecosystem (Metcalf et al., 1973). Approximately 17% of the labeled carbon was recovered in metabolites from a snail, *Physa sp.* The metabolites could not be identified, but one unknown was suggested to be 9-hydroxyendrin by analogy with dieldrin degradation. Lower bioaccumulation factors for the degradation product (1701) compared with endrin (49,000) also support the formation of a hydroxylated compound that is more soluble than the parent molecule (Metcalf et al., 1973). No metabolites were found after the arthropod *Daphnia sp.* was exposed to endrin (Menzie, 1969).

Endrin was eliminated by clams at a rate comparable to that of uptake (Butler, 1971). *Mya arenaria* accumulated 2.6 times (620 ppb) as much endrin as did *Mercenaria mercenaria* (240 ppb) in five days and released 3.1 times (610 ppb) more endrin in seven days of flushing.

The Australian freshwater mussel, *Hyridella australis*, also demonstrated the capability for rapid uptake and elimination of endrin (Ryan et al., 1972). The mussel released 88% of the tissue endrin within 24 h after transfer to fresh water (Fig. 5.1).

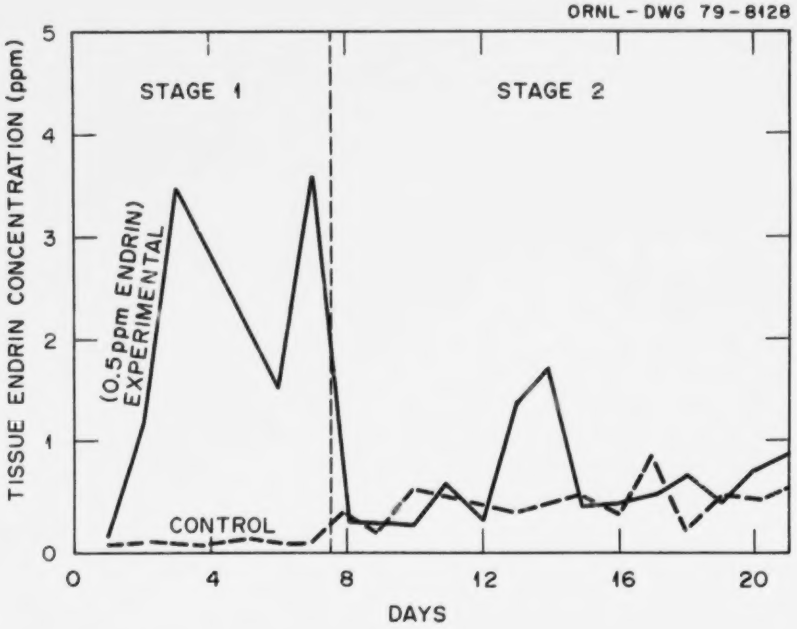

Fig. 5.1. Endrin concentration in mussel tissues after exposure to 0.5 ppm endrin (stage 1) and transfer to fresh water (stage 2). Source: S. Ryan, G. J. Bacher, and A. A. Martin, *Search* 3(11/12): 446-447 (1972). Copyright 1972 Australian and New Zealand Association for the Advancement of Science.

5.2.1.2 Effects. Endrin is more toxic to the aquatic arthropods than to mollusks. Lethal endrin concentrations are less than 10 ppb for several species of marine shrimp, while clams (*M. mercenaria)* and mud snails (*Nassa obsoleta*) survive at levels a thousand times greater (10 ppm) (Eisler, 1970a).

Table 5.2 gives LC_{50} endrin values for some aquatic invertebrates. Effective endrin concentrations decrease with increasing exposure time (Fig. 5.2). Small changes in endrin levels may make the difference between an ineffective concentration and lethality, as is demonstrated by death

Table 5.2. Endrin LC_{50} values for aquatic invertebrates

Species	Exposure (hr)	LC_{50} (ppb)	Reference
Planaria	5	1000	1
(*Dugensia spp.*)			
Blue crab	24	100	2
(*callinectes spaidus*)	48	25	2
Hermit crab	24	2.7	3
(*Pagurus longicarpus*)	96	1.2	4
Freshwater crayfish	3.3	1800	1
(*Cambarus spp.*)			
Red crawfish	48	300	3
(*Procambarus sp.*)			
Grass shrimp	24	1.5	5
(*Palaemonetes pugio*)			
(*P. vulgaris*)	24	10.3	3
	96	10	4
Brown shrimp	24	0.6	2
(*Penaeus aztecus*)	48	0.3	2
Sand shrimp	24	2.8	3
(*Crangdon septum spinosa*)	96	1.7	4
Waterflea	24	900	6
(*Daphnia magna*)	50	352	6
(*D. pulex*)	0.1	1000	1
(*Simocephalus serrulatus*)	48	26	3
(*Moina macrocopa*)	24	3200	7
	48	56	7
Stonefly naiads	96	0.32	8
(*Acroneuria pacifica*)			
(*Pteronarcys californica*)	24	4–24	3
	48	0.9–6	3
	96	2.4	8
Mayfly nymph	48	5	3
(*Baetis sp.*)			
Freshwater isopod	0.5	1000	1
(*Asillus spp.*)			
Freshwater amphipod	2.5	1000	1
(*Gammarus sp.*)	24	8	3
	48	5	3

1. Georgacakis and Khan, 1971.
2. Butler, 1963.
3. Pimentel, 1971.
4. Eisler, 1970a.
5. Hansen et al., 1973.
6. Sanders and Cope, 1966.
7. Iyatomi et al., 1958.
8. Jensen and Gaufin, 1966.

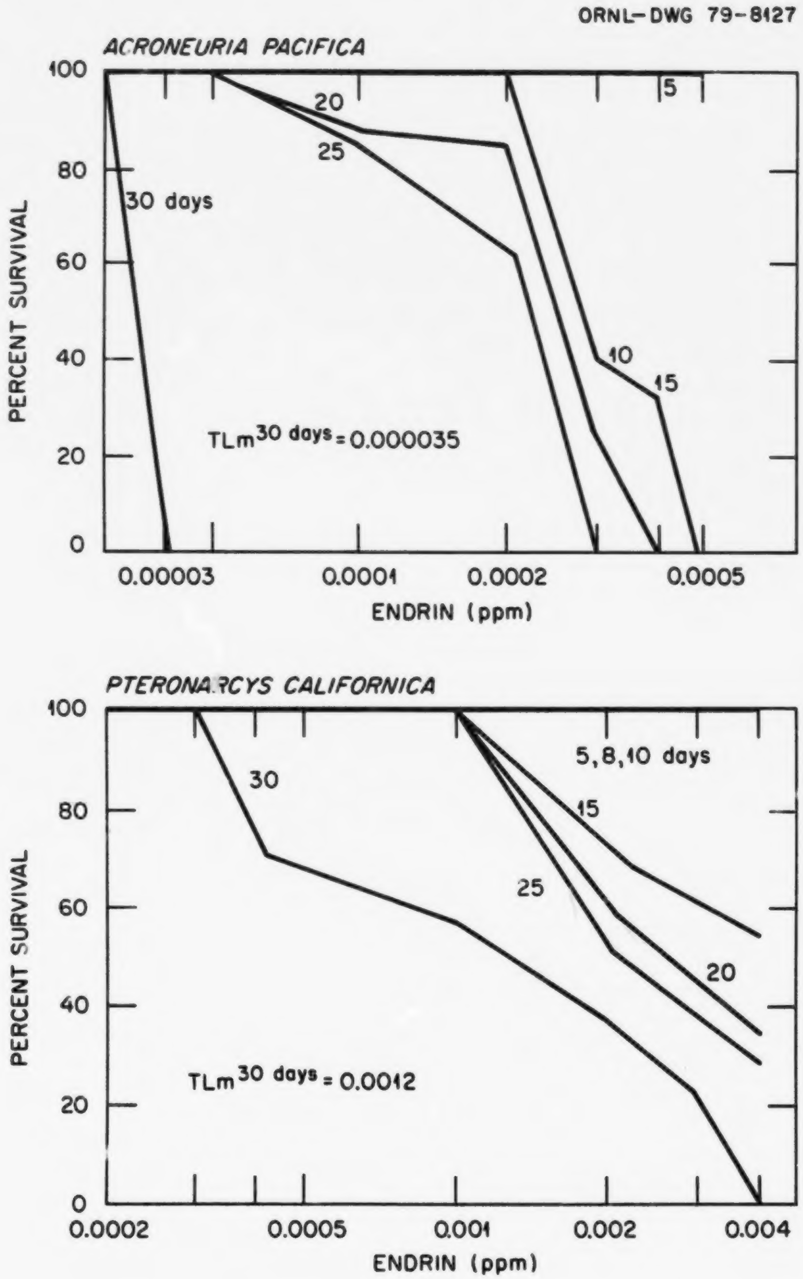

Fig. 5.2. Effects of endrin concentration and exposure time on two stonefly naiad species. Source: L. D. Jensen and R. A. Gaufin, *J. Water Pollu. Control Fed.* 38(8): 1273-1286 (1966). Copyright 1966 Water Pollution Control Federation.

of both water fleas (*Daphnia magna*) and mosquito larvae (*Culex pipiens quinquefasciatus*) when endrin is increased from 10 ppb to 60 ppb (Metcalf et al., 1973).

Evidence of endrin toxicity in oysters (*Crassostrea virginica*) is first manifested by decreasing shell growth, with the percent of inhibition directly related to the concentration (Butler, 1966a, 1963). The percent decrease in shell deposition over 96 hr was 70, 40, 20, and 0 for endrin concentrations of 1.0, 0.1, 0.01, and 0.001 ppm respectively. The sequence of poisoning symptoms in freshwater shrimp (*Palaeomonetes kadiakensis*) and stone fly naiads (*Pteronarcys spp.*) is similar (Sanders and Cope, 1968; Naqvi and Ferguson, 1970; Jensen and Gaufin, 1966). Initially there is hyperactivity, increasing in intensity with exposure time. This is followed by tremors, loss of equilibrium, convulsions (stone fly naiads), paralysis (shrimp), and death. Once tremors and convulsions occurred in two species of stone fly naiads (*Pteronarcys californica* and *Acroneuria pacifica*), there was no recovery (Jensen and Gaufin, 1966). These observations suggest that endrin exerts its toxicity by affecting the nervous system, possibly through action on critical enzyme systems (Jensen and Gaufin, 1966).

Increasing temperature raises endrin lethality to oysters (Butler, 1966a, 1966b) but decreases effects in daphnids (Sanders and Cope, 1966). The juvenile stages of aquatic invertebrates are generally more susceptible to endrin than are adult forms, including oysters (Davis, 1961), daphnids (Sanders and Cope, 1966), red crawfish (*Procambarus sp.*), and stoneflies (Jensen and Gaufin, 1966).

The lethal concentration of endrin may be dependent upon the morphology of a species; for example, the 20-day LC_{50} of the thin-shelled stone fly (*Acroneuria pacifica*) was 0.25 ppb, approximately one-tenth of the level for the hard-shelled stone fly (*Pteronarcys californica sp.*) (Jensen and Gaufin, 1966). Stone fly data demonstrate an interspecific correlation between size and mortality (Sanders and Cope, 1968). Stone fly naiads of *Pteronarcys californica, Claasenia sabulosa,* and *P. badia* (listed in order of decreasing species size) had 24-hr LC_{50} endrin of 4.0, 3.2, and 2.8 ppb respectively.

Sublethal endrin concentrations affect growth and reproduction of many aquatic invertebrates. Survival and growth of oyster larvae (*C. virginica*) exposed to endrin (0.5 to 10 ppm) were erratic, probably due to solubility differences, but larvae were generally more sensitive than eggs (Table 5.3) (Davis, 1961). Increased larval growth at 1.0 ppm may be due to bacteriostatic action. Endrin concentrations (10 ppb) decreasing oyster shell deposition 20% had no observable effect on mortality, spawning, or median growth in mature oysters (Butler, 1971). Endrin reduced the deposition of egg cases by mud snails (N. *obsoleta*) at 0.1 ppm but significantly increased deposition after 33 days of exposure to 0.1 ppm (Eisler, 1970a). Enhancement may not necessarily be beneficial,

Table 5.3. Effects of endrin on survival and growth of oyster (*Crassostrea viginica*) larvae and eggs

Endrin (ppm)	Survival and growth (%)	
	Larvae	Eggs
0.50	36	72[a]
1.00	92	18, 62[a]
2.50	52	4, 60[a]
5.00	10	52[a]
10.00	0	0[a]

[a] Same stock suspension, two weeks later; probably flocculated.

Data from Davis, 1961.

since deposition over longer periods may be a means to avoid loss of an entire reproductive effort. Experiments were not carried out long enough to determine whether increased deposition continued or was the result of a single effort. The molting rate of stone fly naiads was significantly lowered by approximately 3 ppb endrin, decreasing the rate of maturation (Jensen and Gaufin, 1966).

5.2.2 Fish

5.2.2.1 Metabolism

5.2.2.1.1 Uptake and accumulation. Direct sources of endrin to fish include food, water, and sediment. Uptake in fish is based on absorption and solubility differences between fat and water (Hamelink, 1971). Fish act as a multiphase exchange system with successive and reversible partitionings between water, blood, and fat. Thus, endrin is in a dynamic state and not stored in blood or tissue (Mount et al., 1966).

Attainment of lethal endrin levels in the blood of channel catfish is apparently independent of both exposure time and water concentration. In water containing concentrations of endrin that killed some fish but not others, there was a distinct difference between the concentration of endrin in the blood of fish that were killed and those that were exposed but not killed. Figure 5.3 shows some blood levels for fish that survived exposure for 4, 21, and 44 days in water containing endrin concentrations that killed other fish. In 90% of the fish that were analyzed, the endrin concentration in the blood of fish killed by endrin was greater than the concentration of endrin in the blood of fish that were exposed but not killed. No fish survived blood levels exceeding 0.28 μg/g, and no fish died with less than 0.23 μg/g.

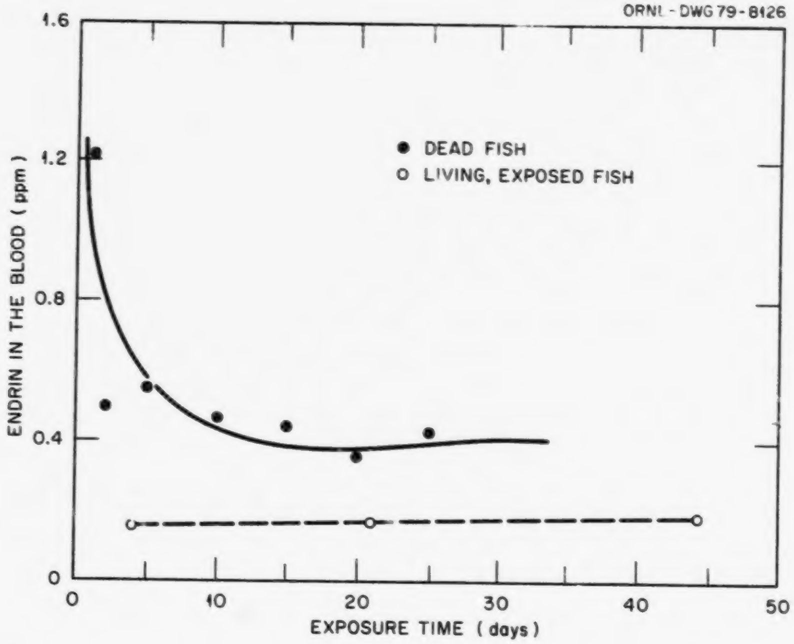

Fig. 5.3. Blood levels of endrin in channel catfish exposed to 0.1 ppb endrin. Source: D. I. Mount et al., *Science* 152(3727): 1388-90 (1966). Copyright 1966 by the American Association for the Advancement of Science.

A continuous mortality of channel catfish, *Ictalurus punctatus*, and brown bullheads, *I. nebulosis*, in Alabama between September 1975 and March 1976 was investigated by Plumb and Richburg (1977). Sera of moribund catfish averaged 0.29 to 1.80 μg endrin/g. After cessation of mortality in March 1976 the sera of apparently healthy catfish from the affected ponds had <0.02 μg endrin/g. During the period of chronic mortality, water samples from the affected ponds had very low concentrations of endrin (0.06 to ≤0.01 μg/liter). They postulated that survivors of a 1975 pesticide kill absorbed insecticides and stored them in fat tissue; the fat reserves were then utilized during a period of decreasing food availability, releasing the endrin and producing toxicosis.

The rate of uptake from food by channel catfish is directly proportional to the concentration (Argyle et al., 1973). After 20 days, whole-body endrin concentrations were 310, 37, 6 ppm and undetectable from exposure to 4, 0.4, 0.04, and 0.004 ppm respectively.

Endrin concentration in water also determines the initial uptake rate and equilibrium levels (Argyl et al., 1973). However, uptake from water far exceeds that from food, bioconcentration from water being 2000 times higher than that from food in channel catfish (Argyl et al., 1973).

The lower bioconcentration from food could result from equilibration in fish, since endrin absorbed from the gut is subsequently released to the endrin-free water. Or uptake rates and/or routes may be less efficient for endrin contained in food. The highest endrin residues in fish were found in the gut, followed by liver, head, gills, heart and spleen, bodies, and eggs (Ferguson et al., 1966).

Several factors suggest that one means of endrin uptake is through the mouth; for example, young fish with nonfunctional mouths are more resistant to endrin than adults (Ferguson and Goodyear, 1967). However, the primary means of endrin entry into the fish body is absorption through gill surfaces. The evidence of primary uptake by the gills includes:

- Similar mortality rates for starved and fed mosquito fish *Gambusia affinis*) exposed to endrin (Ferguson and Goodyear, 1967).
- Mortality rates for black bullheads (*Ictalurus melas*) with ligated esophagi, sham-operated, and controls were nearly identical (Ferguson and Goodyear, 1967).
- Mosquito fish with only the head area exposed to endrin (1 ppm) had higher mortality rates (100% within 31 min) than fish with only the body exposed to 1 ppm endrin (15% mortality and 2.91 ppm average residue) (Ferguson et al., 1966). (The fish were held in a taut rubber membrane, sealed around the body posterior to the pectoral fins. The membrane was stretched across the top of a jar, separating the endrin solution in the jar from the tap water in which it was immersed.)
- Endrin residues in dead mosquito fish exposed to endrin are about equal to those in live fish without the head area exposed (6.25 and 6.34 ppm, respectively, after 11.25 hr in 1 ppm endrin) (Ferguson et al., 1966).

Endrin absorption by bluegills (*Lepomis macrochirus*) produced different curves for lethal (2 ppb) and sublethal (0.2 ppb) exposures (Bennett and Day, 1970). Lethal doses showed increasing body concentrations with increasing time, while sublethal exposures gave an "N-shaped" curve. It appears that, initially, endrin is rapidly absorbed, but after 7 or 8 hr, a "coping" mechanism is developed that may allow metabolism or excretion of endrin, resulting in a concentration drop. A gradual increase in endrin follows, until the contending processes of uptake and excretion reach their respective levels and an equilibrium is attained. Muscle and liver tissue uptake curves are similar to those for the whole body. Gut uptake, on the other hand, showed no drop when bluegills were exposed to either lethal or sublethal concentrations of endrin, probably as a result of steady absorption from the swallowed water.

Endrin-resistant and -susceptible mosquito fish show significant differences in endrin uptake in the brain and nervous tissue, suggesting some sort of membrane barrier preventing or slowing endrin entry (Wells et al., 1972; Wells and Yarbrough, 1973; Fabacher and Chambers, 1976). The ratio of endrin concentration in brain to that in liver was not only higher in susceptible fish (1:30) than in resistant fish (1:8), but total brain endrin was three to four times higher in susceptible fish (Wells and Yarbrough, 1972; Yarbrough, 1974). Cell membrane fractions from brains of endrin-resistant fish bind twice as much endrin as those from susceptible fish, thus reducing the amount of endrin entering the cell (Wells and Yarbrough, 1972). On the other hand, brain fractions other than cell membranes (e.g., microsomal and myelin fractions) manifested more endrin binding in susceptible fish than in resistant. Increased binding of endrin to susceptible myelin fractions may lead to interference with nerve transmission (Wells and Yarbrough, 1972).

Fabacker and Chambers (1976) suggested that at higher concentrations of endrin, mechanisms of resistance may be important in resistant mosquito fish, *Gambusia affinis*, such as a more efficient blood-brain barrier. This may prevent large amounts of endrin from accumulating in the brain, allowing a more rapid accumulation in the lipid stores of the liver. To support this they found that the brains and livers of susceptible fish killed with 20 ppb endrin contained 1.9 times more endrin than the same tissues from resistant fish treated with the same amount of endrin. Fish mortality in both the resistant and susceptible strains occurred with endrin concentrations of 1000 ppb. Brains of the susceptible fish had higher endrin levels, and the livers of the resistant fish had 2.2 times more endrin than those of the susceptible fish.

Endrin uptake was one-third as fast into muscle preparations of resistant nervous tissue (identified as the primary site of endrin action) as into susceptible nerve tissue at low exposure levels (10 ppb) (Yarbrough, 1974). At acute concentrations, the difference in uptake rates (resistant/susceptible) was only one-half, probably as a result of comparing "tolerant" susceptible fish with resistant ones, since only a few susceptible fish survived 1500 ppb endrin.

Whole-body uptake of endrin also decreased in fish previously exposed to endrin (Ludke et al., 1968). Resistant golden shiners accumulate approximately 60% of the endrin taken up by susceptible fish, 260 and 420 ppb, respectively, after 1 hr in 10 ppb endrin.

5.2.2.1.2 Transformation and elimination. The evidence for endrin transformation by fish is based on one investigation by Metcalf et al. (1973). Mosquito fish altered approximately 25% of introduced endrin after 33 days of exposure. Identification of one of the metabolites as a polar compound suggested a hydroxylated degradation product, 9-hydroxyendrin. Speculation of the product's identity was made on the basis of analogy with dieldrin metabolism in rats and a bioaccumulation factor considerably lower (approximately one-fifth) than that for endrin. The amount of endrin calculated to have been transformed into polar products was small, 0.9% (Metcalf et al., 1973).

No evidence of metabolites was found in fathead minnows (*Pimephales promelas*) (Menzie, 1969) or in dead and living mosquito fish by other investigators (Ferguson et al., 1966).

Endrin is released rapidly from fish withdrawn from endrin exposure. Since the amount of endrin released from living mosquito fish is not significantly different from that released by dead ones, it appears that active secretion is not involved (Johnson, 1968; Ferguson et al., 1966). However, active endrin elimination at sublethal concentrations has been demonstrated (Bennett and Day, 1970).

Endrin residues in spot (*Leiostomus xanthurus*) at levels of 78 ppm whole-body weight disappeared within 13 days of endrin withdrawal (Lowe, 1965). Channel catfish lost approximately two-thirds of a 310-ppb residue after 28 days, with no detectable levels after 41 days of an endrin-free diet (Argyl et al., 1973). The rapid decline in endrin residues demonstrates that the compound can be eliminated in a relatively short time.

5.2.2.2 Effects. Endrin is the most toxic to fish of the common organochlorine insecticides (e.g., toxaphene, dieldrin, aldrin) (Eisler, 1970c). Table 5.4 lists some LC_{50} values for a variety of fish, with different exposure times. Because pollutants may drain into aquatic environments for considerable periods of time and at low levels due to the dilution factor, LC_{50} values for 24 and 48 hr have questionable value except to measure relative toxicity (Lowe, 1965; Butler, 1966a). For example, a 24-hr LC_{50} of 1 ppm may not be considered dangerous, because there is little chance of that concentration in the field. However, the same fish may have a ten-day LC_{50} of 0.03 ppb, a very possible degree of pollution. Another factor to be considered in using LC_{50} values is that the margin of safety and lethality for fish exposed to endrin is exceedingly narrow (Pimentel, 1971). Spot (*Leiostomus xanthurus*) flourish at 0.05 ppb, but an increase to 0.1 ppb causes 100% mortality in five days (Butler, 1966a; Pimentel, 1971).

A correlation between concentration of endrin in the blood and mortality in channel catfish (Mount et al., 1966) and gizzard shad (Brungs and Mount, 1967) was found in both laboratory and field studies. Critical blood concentrations were approximately 0.3 and 0.1 ppm endrin for catfish and shad respectively. Some correlation between mortality and whole-body residue is found in shiner perch (*Cymatogaster aggregata*) and dwarf perch (*Micrometrus minimus*), but overlap occurs (Earnest and Benville, 1972). The endrin residues found in dead and live shiner perch averaged 0.13 (0.02 to 0.27) and 0.07 (0.03 to 0.09) ppm respectively.

Many factors influence the effective endrin concentration; among these are: exposure time, temperature, salinity, body size, previous exposure, flowing or static bioassay systems, species, synergism with other compounds, route of endrin uptake, developmental stage, and the season of the year.

The lethality of endrin to carp (*Cyprinus carpio*) and snakehead fish (*Channa argus*) increased during the course of their development (Iyatomi et al., 1958). The fertilized eggs and 1-, 4-, 6-, and 12-day

Table 5.4. Endrin toxicity to some fish as measured by LC_{50}[a]

Family and species	Exposure (hr)	LC_{50} (ppb)	Source
Anguillidae			
American eel (*Anguilla rostrata*)	24	1.1	1
	48	0.6	1
	96	0.6	1, 2
Centratchidae			
Bluegill sunfish (*Lepomis macrochirus*)	24	0.35–2.0	3, 4, 5
	36	1.5	6
	72	0.95	7
	96	0.25–0.6	7, 8
(LD_{50})	6	200	9
	24	10	9
Green sunfish (*L. cyanellus*)	36	3.4–5	6, 10
Clupeidae			
Menhaden — juvenile (*Brevoortia sp.*)	24	0.8	11
Cyprinidae			
Bluntnose minnow (*Pimephales notatus*)	96	0.3	8
Carp (*Cyprinus carpio*)	48	140	8
(LD_{50})	24	5–9	12
	48	5	12
Fathead minnow (*P. promelas*)	1	700	9
	48	0.57	13
	96	0.39–1.8	8, 13, 14
Goldfish (*Carassius auratus*)	24	3	3
(LD_{50})	48	2	3
	96	2	8
Golden shiner (*Notemigonus crysoleucas*)	36	3	6
Cyprinodontidae			
Longnose killifish (*Fundulus similis*)	24	0.3	3
	96	0.3	3
(juvenile)	24	0.23	11
Mummichog (*F. heteroclitus*)	24	1.8	1
	48	0.7	1
	96	0.6	1, 2, 15
	240	0.33	15

[a] No resistant fish used knowingly.

Table 5.4 (continued)

Family and species	Exposure (hr)	LC_{50} (ppb)	Source
Sheepshead minnow — juvenile (*Cyprinodon variegatus*)	24	0.32	11
Striped killifish (*F. majalis*)	24	1.8	1
	48	0.7	1
	96	0.3	1
Embiotocidae			
Dwarf perch (*Micrometrus minimus*)	96	0.6	16
Shiner perch (*Cymatogaster aggregata*)	96	0.8	16
Gasterosteidae			
Three-spined stickleback (*Gasterosteus aculeatus*)	48	0.45–2	7, 14
	72	0.45–1.7	7, 14
	96	0.44–1.5	7, 14
Heteropneustidae			
Indian catfish (*Heteropneustes fossilis*) (LC_{100})	5	120	17
	24	6	17
Ictaluridae			
Black bullhead (*Icatalurus melas*)	24	1.3	3
	36	0.37	6
	96	1.1	3
Channel catfish (*I. punctatus*)	24	0.45	3
	96	0.29	3
Yellow bullhead (*I. natalis*)	36	1.25	18
Larbridae			
Bluehead (*Thalassoma bifasciatum*)	24	0.6	1
	48	0.5	1
	96	0.1	1
Mugilidae			
Striped mullet (*Mugil cephalus*)	24	0.7	1
	48	0.3	1
	96	0.3	1, 19
(juvenile)	24	2.6	11
White mullet — juvenile (*M. curema*)	24	0.3–2.6	3, 20
	96	2.6	3
Ophicephalidae			
Snakehead fish (*Channa punctatus*)	24	8.5	21
	48	4.1	21
	72	2.7	21
	96	1.9	21

Table 5.4 (continued)

Family and species	Exposure (hr)	LC_{50} (ppb)	Source
Percichthyidae			
Striped bass (*Morone saxatilis*)	96	0.09	22, 23
(juvenile)	96	0.94	22, 23
Poecilidae			
Atlantic silversides (*Menidia menidia*)	24	0.5	1
	48	0.08	1
	96	0.05	1, 19
Guppy (*Lebistes reticulatus*)	72	1.1	7
	96	0.3–1.5	7, 8, 14
Mosquito fish (*Gambusia affinis*)	36	1	6
	48	1–1.8	7, 24
	72	1.05	7
	96	0.75	7
Salmonidae			
Brook trout (*Salvelinus fontinalis*)	24	1.28	25
	48	0.59	25
	72	0.42	25
	96	0.36	25
Chinook salmon (*Oncorhyncus tshawytscha*)	24	2	14
	48	1.2–1.5	7, 14
	72	1.01–1.2	7, 14
	96	0.92–1.2	7, 14
Coho salmon (*O. kisutch*)	24	1.3–12.	7, 14
	48	0.56–0.8	7, 14
	72	0.3–0.52	7, 14
	96	0.27–0.51	7, 8, 14
Cutthroat trout (*Salmo clarki*)	48	0.24	25
	72	0.15	25
	96	0.11	25
Rainbow trout (*S. gairdneri*)	24	0.8–5.3	3, 7, 14
	48	0.6–1.5	7, 14
	72	0.5–1.1	7, 14, 25
	96	0.4–1.4	3, 7, 14, 25
Sciaenidae			
Spot (*Leiostomus xanthurus*)	24	0.45	3
(juvenile)	24	0.5–4.4	3, 11
	96	0.6	3

Table 5.4 (continued)

Family and species	Exposure (hr)	LC_{50} (ppb)	Source
Tetraodontidae			
Northern puffer (*Sphaeriodes maculatus*)	24	3.1	1
	48	3.1	1
	96	3.1	1, 2
(LC_{50})	24	10	2

Sources:
1. Eisler, 1970c;
2. Eisler and Edmunds, 1966;
3. Holden, 1973;
4. Cope, 1963;
5. Bennett and Day, 1970;
6. Pimentel, 1971;
7. Katz and Chadwick, 1961;
8. Johnson, 1968;
9. Georgacakis and Khan, 1971;
10. Finley et al., 1970;
11. Lowe, 1965;
12. Iyatomi et al., 1958;
13. Lincer et al., 1970;
14. Katz, 1961;
15. Eisler, 1970b;
16. Earnest and Benville, 1972;
17. Saxena and Aggarwall, 1970;
18. Ferguson and Bingham, 1966b;
19. Duke and Dumas, 1974;
20. Butler, 1963;
21. Javaid and Waiz, 1972;
22. Korn and Earnest, 1974;
23. Suffett et al., 1975;
24. Ferguson, 1967;
25. Post and Schroeder, 1971.

larvae of the snakehead fish had 24-hr median tolerance limits (LD_{50}) to endrin of 100, 62, 5, 6, 0.012, and 0.0065 ppm respectively.

After exposure to sublethal endrin concentrations (0.05 ppb) for eight months, spots were more sensitive to lethal endrin concentrations (0.56 ppb or more) than were unexposed control fish (Lowe, 1965). At levels of 0.32 ppb endrin or less, both exposed and control fish showed similar responses. Mummichogs were able to withstand short immersions in toxic concentrations of endrin (3 ppb, five times the 96-hr LC_{50}) (Eisler, 1970b). After 6 hr, survival rapidly decreased with extended exposure (Fig. 5.4). Posttreatment survival also was correlated to the length of exposure.

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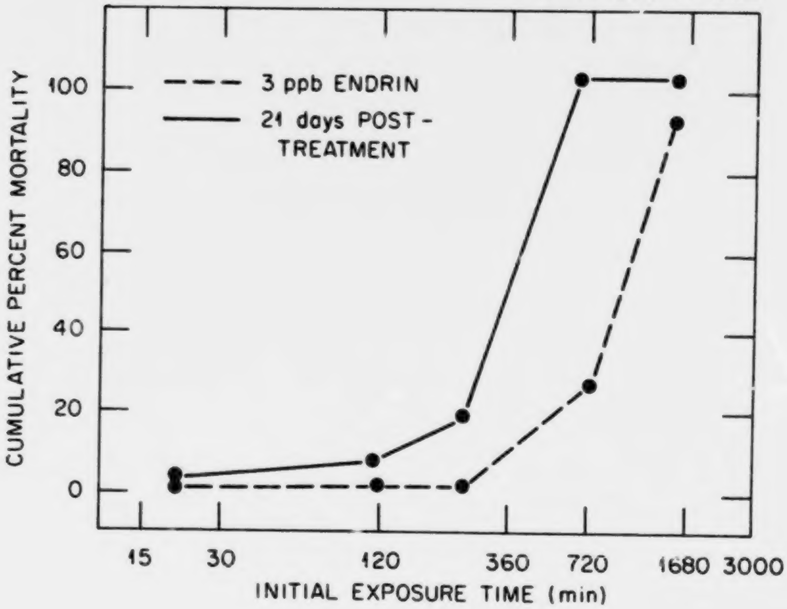

Fig. 5.4. Mortality of mummichogs to various durations of endrin treatment. Source: Eisler, 1970b.

Endrin appears to primarily affect the nervous system of fish. The typical symptoms of endrin poisoning bear out central nervous system damage: hypersensitivity, hyperactivity, loss of sense of direction, loss of equilibrium, increased ventilation, convulsions, and inert (except for operculum moving) on the bottom (Johnson, 1968; Javaid and Waiz, 1972; Saxena and Aggarwall, 1970; Argyl et al., 1973; Eisler and Edmunds, 1966). El-Refai et al. (1976) also found that carp, *Cyprimus carpio*, and tilapia, *Tilapia milotica*, slowly lost neural control of their swim bladders and rose to the surface after exposure to endrin-contaminated water.

The increased activity resulting from endrin exposure may cause the ineffective feeding behavior observed in goldfish (Grant and Mehrle, 1970) and disrupt normal swarming and displacement behavior in guppies (Pimentel, 1971). Sheepshead minnows (*Cyprinodon variegatus*) showed avoidance behavior to endrin concentrations only in the range 1.0 to 0.1 ppb (Hansen, 1969), but mosquito fish did not avoid 10 ppb endrin or less (48-hr LC_{50} = 1 ppb) (Hansen et al., 1972).

Chronic endrin exposures caused reduced reproduction in bluntnose minnows and goldfish (143 μg/kg body weight per day), probably as a result of decreased gonad development (Eisler and Edmunds, 1966; Grant and Mehrle, 1970). Cutthroat trout fed 0.1 ppm endrin daily had irregular oocytes (Eller, 1971), and exposure of guppies to 0.5 ppb prevented reproduction (Eller, 1971). Adult carp at threshold toxicity

(5 ppm) showed some degree of abortion, but egg embryo viability was not affected below 1 ppm (Holden, 1973). Growth was halved in rainbow trout fed 145 ppb endrin per body weight daily (Grant and Mehrle, 1973). Endrin decreased growth in goldfish fed 430 ppb daily but increased growth at 43 ppb or less (Grant and Mehrle, 1973).

Endrin impairs the function of liver tissue (Holden, 1973; Grant and Mehrle, 1970, 1973; Eisler and Edmunds, 1966; Lowe, 1965; Eller, 1971). Eller (1971) reported a hepatitis-like response in cutthroat trout liver, including increased blood coagulation time and degeneration of the liver. The incidence and severity of liver degeneration increased with exposure to increasing endrin concentrations (10 ppb or more). The major liver effects encompassed energy transformations, such as reduction of liver glycogen (Eller, 1971; Lowe, 1965; Grant and Mehrle, 1973). Eller (1971) suggests that endrin and nutritional stress interact to impair the liver and fatty-acid metabolism.

The changes in blood and serum constituents reflect the liver dysfunction. Serum glucose increased 50% over controls (145 ppb), and there was a significant correlation between endrin dose (4.3 to 145 ppb) and both serum sodium and increased osmolality in trout (Grant and Mehrle, 1973). Other serum alterations included increased lactate, decreased amino acids, and increased cortisol. Northern puffer (*Sphaeroides maculatus*) also exemplified cation transfer from liver to serum, 1.0 ppb endrin causing increased sodium, potassium, and calcium in the serum and decreased levels in the liver (Eisler and Edmunds, 1966). Fishes from the Mississippi River fish kills in 1963-64 (generally attributed to endrin) had lowered red and white blood cell counts (Johnson, 1968).

Pathologic conditions were generally found in the gills and pancreas of fish as well as in liver. Gill tissue in trout was edematous as a result of increased osmolality, undoubtedly interfering with gill function and water circulation and contributing to the increased ventilation observed in endrin-poisoned fish (Eller, 1971). Increased cell numbers in the islets of Langerhans were observed in cutthroat and rainbow trout (11 months in 0.01 ppm), but the implied alteration of carbohydrate metabolism was not confirmed by biochemical analysis (Grant and Mehrle, 1973; Eller, 1971). Lesions were present in the brain, spinal cord, liver, kidney, and stomach of spot chronically exposed to endrin (0.075 ppb) (Holden, 1973).

Endrin has been identified as an inhibitor of several enzyme systems, including ATPases (Cutkomp et al., 1971; Davis et al., 1972), oxidases (Staton and Khan, 1972), and succinic dehydrogenase (Yarbrough and Wells, 1971). The inhibition of $Na^{+}K^{+}$ATPase and Mg^{2+}ATPase in bluegill brain and muscle was almost the same, approximately 20% decrease in activity with endrin concentrations of 16 ppm (Cutkomp et al., 1971). Since endrin toxicity (24-hr LC_{50} = 2 ppb) and ATPase inhibition (20% with 16 ppb) do not correlate, Davis et al. (1972) believe that endrin must act on enzymes other than ATPase. Staton and Khan (1972) reported that endrin is a potent inhibitor of mixed-function oxidase from bluegill sunfish but gave no further data.

Evidence of membrane structural change in response to endrin may be demonstrated by inhibition of succinic dehydrogenase, an inseparable part of the inner mitochondrial membrane (Yarbrough and Wells, 1971). Enzyme inhibition only occurred with intact mitochondria from fish susceptible to the toxic effects of endrin. Disruption of the organelle permitted a similar degree of enzyme inhibition in preparations from both resistant and susceptible organisms. Inhibition of succinic dehydrogenase only after the outer membrane is removed demonstrates the existence of an effective, but not absolute, membrane barrier.

Increased lipid content also appears to be an effect of endrin. The whole-body lipid content was greater in fish exposed to endrin (Fabacher and Chambers, 1971). Endrin-resistant, semiresistant (previously removed to endrin-free water), and susceptible fish had lipid contents of 17, 12, and 9% respectively. Resistant fish livers possess much more fat than susceptible ones, but the amount of endrin solubilized in the fat was comparable (approximately 90% of total endrin taken up) (Fabacher and Chambers, 1971). That fish surviving endrin treatment (40 ppb) all had approximately 50% more fat than did the mortalities (Fabacher and Chambers, 1971) suggests that increased fat may provide a dilution factor.

5.3 BIRDS

5.3.1 Metabolism

A wide variety of possible methods for dispersion of endrin to the natural environment have been noted (Sect. 7.2). Endrin in the avian diet can come from fish, plankton, crustaceans, birds, eggs, or water. Therefore, in considering wild birds, it can be assumed that exposure to endrin is nearly inevitable.

The methods of endrin exposure to experimental avians have been varied. The three primary types of exposure have been accomplished by force-feeding contaminated natural foods (Gregory, 1970), dosing ad libitum in a feed premixed with varying concentrations of endrin (Cummings et al., 1967), or injection of endrin solutions into fertilized eggs (Dunachie and Fletcher, 1969). Force-feeding and injection methods ensure equal exposure of the experimentals; however, ad libitum feeding is the most natural procedure.

There appears to be general agreement that endrin is absorbed from the avian gut and stored in various body tissues by both wild and domestic species (Terriere et al., 1959; Reichel et al., 1969a). However, experimental confirmation of this uptake and absorption was not found.

After absorption of endrin, residues have been reported to be distributed in liver, brain, adipose, eggs, breast muscle, and gonadal tissues. Adipose tissues generally contain the highest concentrations (Gregory, 1970; Terriere et al., 1959), while brain tissues usually contain the lowest residues (Reichel et al., 1969a).

Endrin and four other organochlorine pesticides, lindane, dieldrin, DDT, and heptachlor epoxide, were fed in combination to white leghorn hens. Four groups were initiated on basal diets for two weeks, at which time three of these groups were dosed with respective levels of 0.05, 0.15, and 0.45 ppm each of the five compounds in the feed, while the control group continued the basal diet. The birds on contaminated feed were returned to the basal diet after 14 weeks. The residual level of endrin in fat and breast tissues was found to increase initially with a subsequent plateau at concentrations proportional to dose levels (Fig. 5.5). Since there were considerable differences recorded with respect to the amount of abdominal fat found in the hens, it was concluded that the accumulation of endrin in the abdominal fat did not appear to be dependent upon the amount of adipose available as a repository but rather on the level of pesticide in the feed. In this same experiment, it was also determined that liver residues follow the same general pattern as observed in adipose and breast tissues, that is, a sharp initial increase with an approximate plateau thereafter and a decline during the withdrawal period. Because of significant variations in liver pesticide content, graphic representation was not attempted. The liver plateau levels for the 0.45-, 0.15-, and 0.05-ppm dose levels were 0.35, 0.20, and 0.10 ppm, respectively, with no endrin detected in the controls (Cummings et al., 1967).

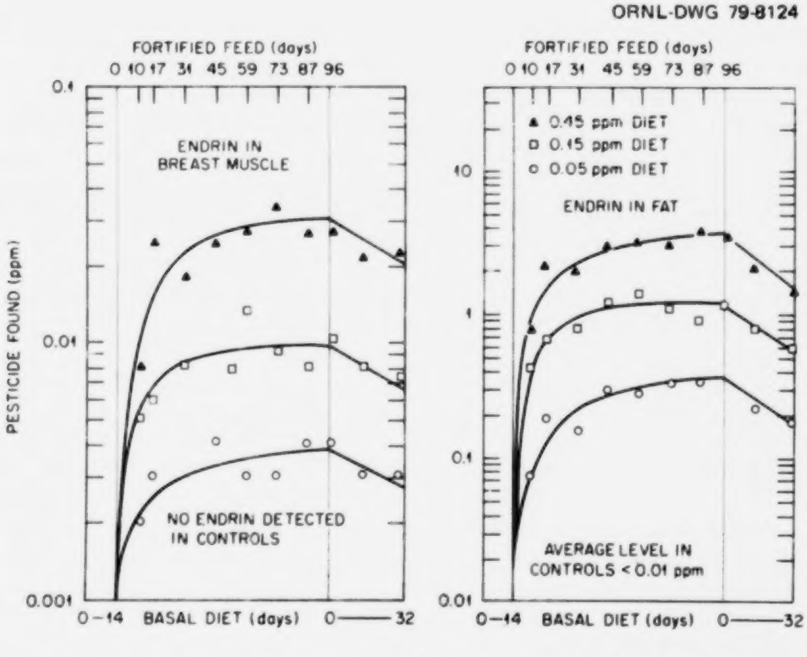

Fig. 5.5. Residual levels of endrin found in breast and fat tissues. Source: J. G. Cummings, M. Eidelman, V. Turner, D. Reed, and K. T. Zee, *J. Assoc. Off. Anal. Chem.* 50(2): 418-425 (1967). Copyright 1967 Association of Official Analytical Chemists.

In a related study conducted by Cummings et al. (1966), residue levels were determined in eggs from hens maintained for 20 weeks on the same low-pesticide-level diets (0.05, 0.15, and 0.45 ppm of each of the five organochlorines). The background level of endrin detected in the eggs before initiation of the pesticide feeding was less than 0.01 ppm. Within three days of exposure to the fortified feed, higher levels were noted. Once again, a direct relationship between plateau level and dosage was observed. The peak residues in the eggs after the hens had been maintained on the diet of 96 days were approximately 0.03, 0.09, and 0.3 ppm for the respective feed levels of 0.05, 0.15, and 0.45 ppm.

Terriere et al. (1959) examined the tissues and eggs of chickens exposed to levels of 0.1, 0.25, and 0.75 ppm endrin in the feed. One-month-old male Delaware X New Hampshire chicks were fed for six weeks and then slaughtered, while six-month-old white leghorn pullets were exposed to the endrin-fortified feed for eight weeks, with a return to the basal diet for four additional weeks. A repeat of the male chick experiment, using New Hampshire X Delaware chicks, was also reported due to analytical discrepancies found in the original testing. In all cases the deposits of adipose were found to be very meager; however, weight gain and feed consumption appeared to be normal for the birds utilized and the experimental conditions imposed. The data accumulated during these experiments are compiled in Table 5.5. At a level of 0.25 ppm or higher, definite deposition of endrin in the egg tissue occurred within two to four weeks after intake. This contamination was still evident for at least one month after exposure had ceased. Accumulation of endrin in adipose tissue was found in both experimental groups, with even the lowest dietary level showing evidence of deposition. Analysis of breast and tibia tissue revealed endrin contamination at both the 0.25- and 0.75-ppm intake levels.

The fat tissue of the plain chachalacas, *Ortalis vetula*, was analyzed by Marion (1976) for pesticide residues during 1971 and 1972. Twenty-four birds, from four different study areas, had an average endrin residue of 0.13 ± 0.52 ppm wet weight. There was no evidence that these birds died of endrin exposure. Only 8 of the 24 birds sampled contained detectable endrin residues.

The percentage of the dose retained by bobwhite quail appears to be dependent upon administration time and dose according to Gregory et al. (1972). Analyses of whole birds fed equal doses of endrin-contaminated beans or beetles revealed retention of approximately 16% of the total acute dose ingested, while 21% of the total chronic dose was retained. The residues found in the body tissues during the acute and chronic experiments differed, but not consistently. The average endrin content of adipose in the acute dosage group was 0.014 ± 0.002 ppm, as compared with 0.010 ± 0.001 ppm found in the chronic dosage group. Gonadal tissues from both groups contained traces of endrin, while liver residues in the chronic test were 0.007 ppm and in the acute test were 0.004 ppm.

Table 5.5. Endrin content of eggs and adipose tissue from white leghorns
(ppm)

Dietary level	During endrin feeding					After endrin feeding cessation					
	1 week		2 weeks	4 weeks		8 weeks		Eggs, 4 weeks		Adipose, 4 weeks	
	A	B	A	A	B	A	B	A	B	A	B
0.00	0.1	0.02	0.1	0.1		0.1		0.1		0.1	0.04
0.10	0.1		0.1	0.1		0.1		0.1		0.1	0.21
0.25	0.1		0.1	0.1	0.05	0.2	0.31	0.1	0.13	0.3	0.59
0.75	0.1		0.1	0.1		0.3	0.36	0.2	0.17	1.1	1.07

Endrin content of broiler tissue from duplicate experiments after 6-week intake
(ppm)

Dietary level	First experiment					Second experiment	
	Adipose		Breast		Tibia,	Adipose	
	A	B	A	B	A	A	B
0.00	0.1	0.02	0.1		0.1	0.1	0.1
0.10	0.5	0.66	0.1	0.04	0.1		
0.25	0.7	1.46	0.1		0.1	0.6	1.0
0.75	1.6	3.10	0.2	0.24	0.3	3.6	4.3
2.25						17.0	18.0

A — analyzed by spectrophotometric method with a detection limit of 0.1 ppm.
B — analyzed by bioassay method with a detection limit of 0.02.

Source: modified from Terriere et al., 1959.

Ludke (1976) fed bobwhite quail (20) diets containing 10 ppm chlordane for a ten-week period followed immediately by 10 ppm endrin for ten weeks. Twenty other quail received 10 ppm endrin only in the diet for nine to ten days. All birds lost weight; mortality from the endrin diet was associated with as little as 0.34 ppm in the brain of the quail.

Evidence for the biotransformation of endrin by avians was not found. Terriere et al. (1959) suggested that, based on estimates of the percent retention of endrin by birds, either some of the endrin is metabolized and stored in a chemical form not detected by the analytical methodology used or that endrin or its metabolites are excreted. Supporting or conflicting evidence for these theories was not found.

The distribution of endrin by egg-laying hens to their eggs, as discussed previously, may be considered a method of elimination; however, it has not been established that egg laying significantly reduces the endrin body burden of the hen.

5.3.2 Effects of Endrin

In the literature, mortality appears as the most frequent effect of endrin. The mean lethal concentrations (LD_{50}) for endrin and a group of 88 other chemicals were established by Heath et al. (1972). Using two- to three-week-old bobwhites, pheasants, mallards, and Japanese quail at the Patuxent Wildlife Research Center in Laurel, Maryland, the LC_{50} and RTD (relative toxicity to dieldrin) were derived by the method of probit analysis. A dieldrin standard was consistently used to adjust for possible experimental differences in animal sensitivity or physiological and environmental variations. The selection of dieldrin was based on its ability to provide an acceptable probit regression line to facilitate comparison of experimental data. This investigation attempted to measure the relative toxicities of each of the 89 chemicals on the basis of the data compiled. Endrin ranked as one, or the most toxic chemical tested, in all four of the experimental avian species (Table 5.6).

A 30-day empirical minimum lethal dosage (EMLD), the lowest daily oral dose that produces one or two deaths by the end of the 30-day period, of 0.125 mg endrin/kg/day was established for mallards. Since the LD_{50} was 5.64 mg/kg in this experiment, it was calculated that the cumulative toxicity index of endrin was 45 (5.64/0.125), indicating a moderately high degree of cumulative action (Tucker and Crabtree, 1970).

A comprehensive study of the toxicity of endrin to quail and pheasants, both adults and juveniles, was conducted by DeWitt (1955). Severe tremors, muscular incoordination, and extreme nervousness ensued in adult quail within 2 hr of administration of a diet containing 0.50%, or 5000 ppm, endrin. All birds in this group died within 48 hr. It was noted that lower dietary concentrations resulted in a lethargic condition and bedraggled plumage prior to the onset of these symptoms of acute toxicity. Endrin also proved highly toxic to adult pheasants. Diets containing 0.025%, or 250 ppm, were refused by the birds, but satisfactory

Table 5.6. Comparative dietary toxicity of endrin to birds

Species	No. of concentrations	Birds per concentration	LC_{50}: ppm chemical in feed[a]	95% confidence limits	Relative toxicity of dieldrin (RTD)[b]	95% confidence limits
Bobwhite	6	10	14	11–24	0.370	c
Japanese quail	6	13	18	15–20	0.304	0.257–0.359
Pheasant	4	8	14	11–17	0.335	0.236–0.527
Mallard	6	10	22	17–31	0.108	c

[a] LC_{50}:ppm endrin in ad libitum diet expected to produce 50% mortality in eight days, five days of toxic diet followed by three days of untreated diet.

[b] RTD reads: "Dieldrin is x times as toxic as the given chemical as tested."

[c] Relative toxicity of dieldrin applies only at LC_{50}, since probit slope is significantly different from that of dieldrin.

Source: Reprinted with permission from J. B. DeWitt, *J. Agric. Food Chem.* 3(8): 672-676 (1955). Copyright 1955 American Chemical Society.

acceptance occurred at concentrations of 0.01%, or 100 ppm. All male pheasants died within ten days of initiation of the toxic diet, while females survived for longer periods (Table 5.7). Continuous feeding of endrin-fortified diets at a level of 0.005%, or 5 ppm, to juvenile quail and pheasants proved to be lethal, and short-time exposure to even lower endrin levels produced a high mortality rate in juvenile quail (Tables 5.8 and 5.9).

Additional studies using quail and pheasant chicks reared on endrin-fortified feed have been attempted; however, a maximum level allowing growth and survival of the chicks was not established (DeWitt, 1956). Within 48 to 72 hr of initial feeding of 20 ppm endrin, symptoms of acute poisoning appeared, and food consumption was significantly reduced during the remainder of the test. Muscular incoordination, bedraggled appearance, occasional tremors, and stiff-legged, hesitant movements were followed by spasmodic leaping into the air and violent cartwheels which could last for several minutes. Lower levels of endrin delayed the onset of these symptoms; however, growth was depressed in the chicks, and onset of acute poisoning symptoms was usually followed by death within five days. Ataxia, drowsiness, and prostration have also been reported in endrin-poisoned avians (Tucker and Crabtree, 1970).

Other symptoms noted in conjunction with endrin toxicity in chickens include accelerated respiration followed by respiratory difficulty, loss of equilibrium, and convulsive seizures before death. Postmortem investigation has revealed congestion of all internal organs; hemorrhagic areas in serous membranes, epicardium, and endocardium; edematous lungs; and dilated heart (Christopher, 1969).

Severely poisoned pigeons exhibited tonic-clonic convulsions beginning 20 to 30 min after intravenous endrin injection. Within 1 hr of the initial seizure, death usually occurred. However, surviving birds recovered completely and exhibited no detectable symptomatology 4 hr after the injection (Revzin, 1966).

An experimental determination of the toxicity of endrin to New Hampshire chicks is represented in Table 5.10. The dosages listed were administered in one capsule, and results were tabulated seven days later. The deaths occurred between 6 hr and five days of treatment, and acute toxicity symptoms were similar to those previously discussed. Although the effect of treatment on weight gain was evaluated, no significant difference could be determined (Sherman and Rosenberg, 1953).

General conclusions, derived from the experimental data, that may or may not be applicable to all birds include:

1. There appears to be an age-mediated susceptibility difference, in that juvenile avians more readily succumb to the toxicity of endrin than do adults (Sherman and Rosenberg, 1953; DeWitt, 1955).

Table 5.7. Toxicity of endrin to adult quail and pheasants

Species	No. of birds[a]	Concentration fed		Toxicant consumed (mg/kg)		Mortality (%)	Survival time (days)
		%	ppm	Daily	Total		
Quail	10	0.50	5000	62.9	92.5	100	2
	10	0.25	2500	7.2	13.9	100	2
	10	0.125	1250	2.6	9.2	100	4
	10	0.0625	625	1.8	4.5	100	3
	10	0.010	100	0.9	4.4	100	5
	10	0.005	50	0.6	2.8	100	5
	10	0.001	10	0.7	16.7	100	26
	10	0.0005	5	0.5	11.3	100	22
	10	0.0002	2	0.2	6.4	100	36
Pheasants							
Male	5	0.010	100	0.6	5.0	100	9
Female	5	0.010	100	1.1	25.3	100	23

[a] Equal numbers of males and females were used.

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Table 5.8. Toxicity of endrin to juvenile quail and pheasants

Species	No. of birds	Age at start (days)	Duration of test (days)	Concentration in feed (%)	Toxicant consumed (mg/kg)		Mortality (%)
					Daily	Total	
Quail	10	1	2	0.005	1.88	3.2	100
	10	1	5	0.002	1.96	10.0	100
	10	1	6	0.001	1.35	6.9	100
	20	16	19	0.0005	0.38	6.7	100
	20	1	7	0.0005	0.40	2.7	55.0
	32	1	14	0.0001	0.12	1.7	21.9
	22	1	120	0.00005	0.06	7.2	13.6
Pheasants	20	1	5	0.0005	0.36	2.0	100

Source: Reprinted with permission from J. B. DeWitt, *J. Agric. Food Chem.* 3(8): 672-676 (1955). Copyright 1955 American Chemical Society.

Table 5.9. Toxicity of endrin to juvenile quail; intermittent feeding, 28 days between tests

Concentration in feed (%)	Duration (days)	Toxicant consumed (mg/kg)		Mortality (%)
		Daily	Total	
		Initial feeding		
0.001	7	1.35	9.4	81.7
0.0005	7	0.38	2.7	55.2
0.00001	14	0.12	1.7	20.7
		Second feeding		
0.001	7	1.20	6.1	100
0.0005	7	0.19	1.3	69.7
0.00001	14	0.09	1.3	26.0

Source: Reprinted with permission from J. B. DeWitt, *J. Agric. Food Chem.* 3(8): 672-676 (1955). Copyright 1955 American Chemical Society.

Table 5.10. Toxicity of endrin to New Hampshire chicks of various ages

Dosage (mg/kg body weight)	Number	Mortality (%)
	7 days old	
4.3	10	90
3.8	10	90
3.6	10	70
3.3	10	30
2.5	10	10
1.9	10	10
1.4	10	10
Untreated	19	0
	21 days old	
4.3	10	50
3.4	10	0
	45 days old	
4.3	10	40
3.4	20	0
	64 days old	
4.3	10	10
3.4	10	0

Source: C. H. Brett and T. G. Bowery, *J. Econ. Entomol.* 51(6): 818-821 (1958). Copyright 1958 Entomological Society of America.

2. A sex-mediated susceptibility variation occurs in adults birds, since male pheasants died significantly earlier than females, although no eggs were produced by the females (DeWitt, 1955).

3. Lowered feed consumption occurs in birds exposed to endrin-fortified diets, which may result in prolonged survival times before death (Sherman and Rosenberg, 1953; DeWitt, 1955).

Toxicity studies on wild birds are limited due to the difficulties encountered in controlling the imposed conditions. Studies of fields treated with 0.8 lb endrin per acre revealed five dead cackling geese, one dead killdeer, a dead long-eared owl, a dead pheasant, and a dying pheasant. In an attempt to study the effect of endrin on wildlife in a more controlled situation, geese, pintails, widgeion, and pheasants were placed in cages on a field that had been treated as previously described. Within one week, four of eight geese, two of seven pintails, and one of seven widgeon had died, while controls in untreated lots all survived. The pheasants were not adversely affected for the first three days, after which time the birds escaped. Endrin residues found in the tissues of surviving birds were comparable to the levels detected in both the wild and captive birds dying on the same field. Experimental data compiled using widgeon revealed that a daily intake of 1.0 mg/kg would be hazardous or fatal. A 500-g widgeon eating 25 g alfalfa contaminated with 20 ppm endrin, a conservative part of the diet, will ingest 1.0 mg/kg daily (Keith, 1963).

In determination of the hazards of endrin, the reproductive processes are often studied. Egg production, fertility, hatchability, and viability of chicks are the various stages of the avian reproductive process that are often adversely affected by toxicants. Of these four phases, fertility is the only one apparently not altered by endrin exposure.

DeWitt (1956) found that 10 ppm endrin administered in the diet during the reproductive period significantly decreased egg production in pheasants. The other concentrations tested, 2, 1, and 0.5 ppm, did not appreciably alter egg production or any of the other previously mentioned parameters. Fertility of the eggs laid was not decreased by the endrin-fortified diet in either quail or pheasants, and the quail did not experience a decrease in egg production at 1.0 ppm, the highest level administered (Table 5.11).

Hatchability was significantly lowered in pheasants receiving 10 ppm endrin in their feed during the reproductive period. However, lower concentrations did not substantially affect hatchability (DeWitt, 1956). Quail receiving 1.0 ppm endrin in their diet during the winter months and subsequently transferred to an endrin-free food source during the reproductive period also suffered decreased egg hatchability. Reversal of the time of endrin exposure, that is, during the reproductive period rather than during winter, did not appear to reduce hatchability, egg production, or fertility in the quail (Table 5.11) (DeWitt, 1956).

Table 5.11. Effects of endrin upon reproduction

ppm in diet		Number of birds	Mortality (%)	Eggs/hen, average	Fertility (%)	Hatchability (%)	Hatch survival (%)	
Winter period	Reproductive period						2 weeks	6 weeks
				Quail				
		32	6.25	52	89.0	83.9	88.9	83.3
1.0	0	16	25.0	45	84.9	70.1	80.8	50.0
1.0	1.0	10	60.0					
0	1.0	16	25.0	60	93.1	79.0	89.2	64.3
				Pheasants				
		128	0	48	86.6	57.4	94.8	89.7
0	10	10	100	11	81.7	40.6	37.5	31.3
0	2	10	0	42	89.9	47.7	97.1	91.2
0	1	10	0	45	92.6	56.6	91.7	91.7
0	0.5	10	0	40	84.9	71.3	80.0	71.4

Source: modified from DeWitt, 1956.

These same quail, maintained on an endrin-free during the winter and transferred to endrin-fortified feed during the reproductive period, did evidence a reduction in the viability of the hatched chicks, as did chicks from quail fed endrin only during the winter. Pheasant chicks, from parents receiving 10 ppm endrin during the breeding season, also had unusually high mortality rates during the first two weeks. High mortality occurred among quail receiving 1 ppm endrin during both winter and reproductive periods, and, as a result, this group was eliminated from further experimentation (DeWitt, 1956).

Previous experimental investigation using breeding quail fed an 0.001%, or 10 ppm, endrin-fortified diet revealed no visible signs of toxicity as well as no affection of egg production, fertility, hatchability, or viability until 23 days after the onset of the test. All of the adults died within the next three days (DeWitt, 1955).

Other experimental investigations have been conducted on the hatchability and viability of eggs, but they differ from the previous studies in that they utilize egg injection techniques to expose the embryo rather than transfer of endrin from the parents.

Injection of 5 mg endrin/egg, or 100 ppm, resulted in only 20% hatchability compared with 100% hatchability of eggs injected with DDT at the same concentration. A reduction in concentration to 0.5 mg/egg, or 10 ppm, increased the hatchability to 40% (Dunachie and Fletcher, 1966).

Later investigation by Dunachie and Fletcher (1969) revealed that the type of diluent used for endrin could alter the toxicity. Acetone-dissolved endrin was found to be significantly more potent than corn oil-dissolved endrin. Table 5.12 indicates this variation as well as the inconsistency of endrin toxicity to hens' eggs. These inconsistent results were attributed to the possibility that the terminal stage is the most susceptible and that a slight increase in the rigor of the conditions of incubation could significantly reduce hatchability of the treated eggs while not affecting the controls.

Table 5.12. Hatchability of hens' eggs after endrin injection (percentage of control)

ppm	Dissolved in acetone	Dissolved in corn oil
100	30	
50	39	0
25	23	
10	103	46
5	109	

Source: modified from Dunachie and Fletcher, 1969.

In a starvation experiment, the chicks hatched from eggs receiving 5 ppm endrin all died within five days, while the same chicks that received feed after hatching from the eggs injected with endrin survived (Dunachie and Fletcher, 1969). It was felt that this supported the theory that rigorous conditions could adversely affect endrin-exposed avians.

Dunachie and Fletcher (1969) also found that a delay in the time of injection greatly reduced the toxicity of endrin to the embryo. Injection on day 5 of 100 ppm endrin resulted in only 4% hatchability. The same dose given on day 8 resulted in 25% hatchability, while injection on the tenth day did not adversely affect the hatchability of the eggs. These results may indicate that endrin toxicity is altered by either the age of the exposed embryo or the duration of exposure.

Smith et al. (1970) found endrin to be highly toxic to the developing embryo. Only 40% hatchability occurred in eggs injected with 0.2 mg/egg, or 4 ppm, while the control eggs injected with diluent were hatched at a rate of 86.2%. Although the hatchability was greatly affected, there was no physiological effect on the day-old chicks.

Although these injection studies are pertinent, the effect of injection of endrin into the egg may not be similar to the effect of ingestion of endrin by the hen followed by deposition of endrin during formation of the egg.

Endrin is thought to primarily induce central nervous system (CNS) disorder, since behavioral observations indicate that death from experimental endrin poisoning is preceded by convulsions and other CNS-controlled responses. In order to obtain a more detailed knowledge of the action of endrin on the CNS, the telencephalic function of the endrin-exposed pigeon was investigated (Revzin, 1966).

Experiments conducted to determine whether endrin induces a selective or a general pattern of CNS stimulation revealed evidence for both possibilities. All telencephalic nuclei examined, after injection of endrin doses in excess of 4 mg/kg, were induced to seizure with no clear propagation from one center to another. Therefore, endrin at these doses may be considered a nonspecific convulsant. In lower doses, however, the seizures are not identical in all nuclei. The amplitude of the seizure discharges is greater in the ectostriatum and adjacent neostriatum than in the surface leads, paleostriatum or hyperstriatum. Buildup of the high-frequency activity which precedes seizure as well as the initiation of seizure discharge often occurred in the ectostriatum prior to their appearance in other nuclei. Longer duration of the seizure discharges appears to occur in the ectostriatum, and occasionally seizure activity was only measurable in the ectostriatum. These findings, accompanied by the absence of effect induced by endrin on the ascending reticular activating system or on the nucleus rotundus, indicate that endrin also acts as a selective toxicant in the avian CNS.

Resulting from the facilitation of the rotundo-ectostriatal potential after subconvulsive doses of endrin and the knowledge that the ectostriatum is a visual projection area, it is probable that visual deficiencies will result from endrin exposure. These visual deficiencies could reduce the avian ability to avoid predators and compete for food (Revzin, 1966).

Avoidance behavior in the gallinaceous chick occurs early and is of obvious survival value in the wild. Measurement of this avoidance response was tested in seven-day-old Coturnix quail treated with a 2-ppm endrin-fortified diet for eight days and then with untreated feed for six days. Group avoidance response of endrin-treated chicks was significantly suppressed below the controls; however, after 48 hr on an endrin-free diet the birds' avoidance behavior returned to normal (Kreitzer and Heinz, 1974). This alteration of avoidance response coupled with the previously discussed visual deficiencies could greatly reduce the possibility for survival.

A possible synergistic relationship between chlordane and endrin given to Japanese quail was investigated by Kreitzer and Spann (1973). Chlordane and endrin were given in respective mixtures of 80:20, 50:50, and 20:80. None of these doses affected more than an additive mortality in the birds. Therefore, the joint action of chlordane and endrin is neither synergistic nor antagonistic.

5.4 INSECTS

5.4.1 Metabolism

Brooks (1960) treated different strains of houseflies (*Musa domestica*) with ^{14}C-labeled isodrin, aldrin, endrin, and dieldrin in order to determine the metabolism as well as the mechanism of resistance of the adult flies to these insecticides. The radioactive compounds were topically applied to the dorsal thorax of pesticide-sensitive or dieldrin-resistant strains.

The results of this study indicated that insect resistance is not related to the degree of penetration, and that there were no marked differences in the way the radioactive insecticides were metabolized by the different housefly strains. Thus, all strains enzymatically converted isodrin and aldrin to the corresponding epoxides endrin and dieldrin. There was some evidence to show that this enzymatic oxidation took place not only internally, but also occurred on or within the insect cuticle.

Acetone extracts of live susceptible and resistant insects treated with isodrin or endrin contained similar small amounts of water-insoluble products, one of which chromatographically behaved as the ketone, a known rearrangement product of endrin (Bellin, 1956). Endrin and its precursor, isodrin, were found together in all sampled tissues at the time of "knockdown"; however, there was no evidence of endrin excretion by the houseflies.

A more systematic study concerned with the mode of entry, absorption, metabolism, and excretion of endrin by susceptible (S) and resistant (R) larvae was performed by Polles and Vinson, 1972.

In order to determine the mode of entry and the rate of cuticular penetration of endrin, laboratory-reared third instar tobacco budworm larvae (*Heliothis virescens Fabrieius*) and isolated tobacco budworm cuticles were topically treated with ^{14}C-labeled endrin on the dorsal surface (between first and second abdominal segments). Samples analyzed 0, 1, 2, 4, 12, 24, and 48 hr after treatment revealed that penetration of endrin into the endocuticle was slow, and that the rate of penetration was higher in S larvae than in R larvae; however, no specific reason for this difference was elucidated. According to Brooks (1960), the slower penetration into the R strain may be due to the presence of inbred insecticide in the tissues of these insects.

The metabolism of endrin and its distribution within insect tissues was investigated by injecting large amounts [1.25 μg endrin per microliter (0.5 μg ^{14}C-labeled endrin per microliter)] of radioactive endrin in the larvae. The concentration of endrin was then determined in the head, digestive tract, hemolymph, feces, ventral chord, and remaining tissues.

No differences were noted in the localization of endrin in the tissues under study. In both strains the largest amount of endrin was recovered from the fraction termed "remaining tissues" (largely composed of body fat); however, storage of endrin in these tissues appeared to be of no importance as a resistance mechanism, since both S and R strains stored approximately the same amounts of endrin. There was also no discernable strain difference in the accumulation of endrin in nervous system tissue. Differential extraction of nerve tissues with n-hexane and chloroform-methanol solvent systems indicated some degree of affinity of the insecticide for components of the nerve tissue as shown in Table 5.13.

Table 5.13. Quantity of ^{14}C-endrin in S and R larvae of *Heliothis virescens* central nervous system extracted with n-hexane and chloroform-methanol

Extraction solvent	^{14}C-endrin recovered at post-injection[a] (%)					
	Susceptible (S)[b]			Resistant (R)[b]		
	1	6	24	1	6	24
n-Hexane[c]	94.0	88.0	62.0	84.0	66.0	30.0
$CHCL_3$-CH_3OH[d]	6.0	12.0	38.0	16.0	34.0	70.0

[a] Percentage represents total amount in nerve cord.

[b] Hours.

[c] Lightly bound endrin.

[d] Tightly bound endrin.

Source: Reprinted with permission from S. G. Polles and S. B. Vinson, *J. Agric. Food Chem.* 20(1): 38-41 (1972). Copyright 1972 American Chemical Society.

From this data it was concluded that endrin forms complexes with proteins in the nerves of R-strain larvae which are not involved in toxicity. This indicated that the binding of endrin to proteins may result in decreasing the concentration of insecticide available to exert its toxic effits toxic effect. Whether this phenomenon is of significance in the ability of the insect to withstand larger concentrations of endrin is still uncertain.

The results of the metabolic study illustrated that both strains (S and R larvae) produced two metabolites, each tentatively identified as endrin aldehyde and endrin ketone. No evidence of endrin alcohol formation by either strain was found. R-strain larvae produced two additional unidentified metabolites having well-defined R_f values on thin-layer chromatographic plates. Finally, most of the radioactivity excreted by both strains was unchanged endrin.

In an attempt to elucidate the role of insecticide penetration into the insect body as one of the factors affecting the mechanism of insect resistance to pesticides, Szeicz et al. (1973) studied the absorption rate of endrin and other ^{14}C-labeled insecticides into two strains of tobacco budworm, one a susceptible strain, MS, and one an organophosphate resistant strain, CM. Each strain was topically treated with 1 µl of an acetone solution of ^{14}C-labeled endrin. The dose was applied on the dorsal surface of the first abdominal segment. The data obtained in this experiment indicated a higher penetration rate for the more polar materials into larvae of both strains. Thus, approximately 40 to 50% of a 0.2-µg dose of endrin was absorbed within 12 hr by both larval strains. The initial buildup of ^{14}C-labeled insecticide in the cuticle stopped 4 to 8 hr after application. The cuticular accumulation was followed by a similar elevation of the concentration of endrin in the internal fraction. In general the concentration of endrin in the internal fraction exceeded the amount of endrin found in the cuticle after about 6 to 8 hr. The times for maximum internal absorption of endrin were approximately the same for both strains; however, the concentration of endrin at those particular times was greater in the MS larvae. The reason for the measured difference in the rate of penetration in the two strains of larvae used was not discerned, but the results obtained seemed to correlate with those of Polles and Vinson (1972).

5.4.2 Effects

The use of any pesticide for the control of any pest is based on its chemical selectivity for that particular pest. Some insects, however, develop resistance to pesticides; for example, the ability of the housefly to develop resistant strains to DDT was shown by Perry and Hoskins (1951). Perry suggested that insect resistance to DDT was due to the development of detoxification mechanisms.

Mahar (1962) and Eldefrawi et al. (1964) reported the appearance of highly cyclodiene-tolerant strains of the cotton leafworm (*Spodoptera littoralis boise*). Later, Hassan et al. (1970) followed the development of endrin tolerance by different field strains of the cotton leafworm in the laboratory. These investigations suggested that insecticide tolerance was a function of the length of exposure of the worms to the chemical. This was evidenced during 1961 by the inability of the endrin concentrations used to effect an LD$_{50}$. The cessation of the use of endrin during the years 1963 through 1965 caused a decrease of endrin resistance by the worms; however, in 1965, when endrin was once again liberally used, most of the worm strains investigated regained their high tolerance to endrin by the following year.

Attia (1976) found endrin resistance in three strains of the almond moth, *Cadra cantella* (Walker), collected from stored foodstuffs and milk products in New South Wales in 1975. Their endrin response was compared with a human susceptible laboratory reference strain; the field moths exhibited 35-, 57-, and 62-fold greater resistance to endrin.

The mutagenic potential of endrin was studied by Benes and Scram (1969). These investigators used the fruit fly (*Drosophila melanogaster*) as the test organism. The Muller-5 genetic test, which detects recessive lethality in the X-chromosome, as well as the eucentric-chromosome aberration test, which ascertains the rearrangement of larger chromosome groups, showed that endrin did not have any outstanding mutagenic activity in *Drosophila*.

The effects of sublethal treatments of endrin on the biotic potential (ability of a population to reproduce and survive, i. e., natality vs mortality) of lady beetles (*Coleomegilla maculata De Geer*) were investigated by Atallah and Newsom (1966). The reproductive potential (natality rate) was measured by recording the number of eggs produced by each female until death, while the effect of the treatment on the survival potential (ability of the organism to withstand environmental resistance) of the progeny was determined by rearing the F$_1$ larvae and recording the number reaching the adult stage. The results of these studies showed that endrin was toxic to both diapausing and active beetles. The LD$_{50}$ values found were 5.8 μg/active beetle (472 mg/kg) and 5.6 μg/diapausing beetle (455 mg/kg). Although endrin decreased longevity, it had no effect on oviposition or survival of the F$_1$ generation.

Somewhat similar results were reported by Singh and Lal (1966), who studied the biology of red cotton bugs (*D. koenigii*) surviving DDT or endrin treatments. Their findings showed that endrin at concentrations higher than the LD$_{50}$ for the species not only reduced the life span of adult bugs, but also reduced the fecundity of the females and the viability of the eggs laid by the treated parents. On the other hand, lower dosages of endrin (concentrations below the LD$_{50}$) had no effect on oviposition and postoviposition periods. The number of egg batches per female was not affected, and there was no deleterious effect on the first and second generations as compared with controls. One of the most significant findings by Singh and his co-workers was that the female progeny of endrin-treated adults were significantly more tolerant to endrin than the controls and that lower endrin dosages may be responsible for an increased fecundity in the parents' generation.

The susceptibility variations of an insect to insecticides is largely governed by the intrinsic changes in the environmental factors. Of these factors, the quality and quantity of food significantly influence the resistance of insects to insecticides (Gordon, 1961). Abhyankar and Sanup (1970) studied endrin susceptibility of aphids (*Lipaplus erisimi Kalt*) reared on different host plants (six host plants of the *cruciferae* family). Abhyankar's results showed statistically significant differences in the susceptibility of certain groups of aphids to 11 insecticides: phosphamidon, dimethoate ethyl parathion, methyl parathion, malathion, endosulfan, lindane, nicotine sulfate, ppDDT, carbaryl, and nicotine. However, the variations in the endrin susceptibility of aphids reared on these host plants were not statistically significant, leading to the conclusion that the same concentration of endrin can probably be used for the control of aphids irrespective of the host plant. Because there were indications of endrin resistance developed by the aphids, the authors suggested annual evaluations of LD_{50} values.

The antagonistic effect of gossypol, a pigment present in the seeds of glanded varieties of cotton, was studied by Abou-Donia et al. (1974). Acetone solutions of gossypol or acetone alone were deposited on the dorsal regions of army worms (*Spodoptera littoralis*) by means of micropipettes, and the treated larvae were used for the determination of LD_{50} values for endrin and other insecticides. Endrin LD_{50}s increased 200% in the gossypol-treated larvae as compared with the LD_{50} obtained with the non-gossypol-treated controls. Two mechanisms were suggested for the mode of antagonistic action induced by gossypol: (1) gossypol may reduce body penetration of the insecticide; (2) gossypol may enhance detoxification of the absorbed insecticide. The second assumption is supported by findings that liver microsomal oxidases increased in gossypol-fed rats (Abou-Donia and Dieckert, 1971). However, Shaver and Wolfenbarger (1976) applied endrin (25 ug/larva) topically to tobacco budworm, *Heliothis virescens* (F.), larvae and placed them on diets containing 0.07 to 0.0082% (wt/vol basis) gossypol. They determined that the gossypol did not affect endrin's toxicity at this rate.

The mode of toxicity as well as the site of action of the cyclodiene insecticides has not yet been defined. Based on some earlier investigations reviewed by Winteringham and Lewis (1959), it was generally agreed that cyclodienes affect the central nervous system. In an effort to elucidate whether the site of action of endrin is in the ganglia or the axonal membrane, Ryan and Shankland (1971) studied the synergistic action of cyclodiene insecticides with DDT on the membrane and giant axons of the American cockroach (*Piriplaneta americana L.*). From this study it was concluded that the cyclodiene insecticides had no toxic action on the axonal membrane of cockroach giant fibers located in the central nervous system, but did affect the ganglia, suggesting that they are the primary site of pesticide action. It was also found that DDT-cyclodiene mixtures had a joint action on the axonal membrane of the giant fibers of the American cockroach, but this effect did not represent enhancement of the toxicity of either compound.

Although it appears that endrin acts on the central nervous system, the precise mechanism of action on other systems is not understood. In many toxicological studies, insects are used as the test organisms; therefore, a better understanding of the changes in metabolic processes taking place within the test organisms should lead to the selection of those materials ideally suited for their specific control. Following this line of reasoning, Dikshith and Vasuki (1970) studied the changes in phosphatase activity of mealybugs (*Dactylopus confusus CK11*), a large group of insect pests affecting potatoes.

The acid and alkaline phosphatases of the coccid (*D. confusus*) homogenate showed a specificity toward glucose-1-phosphate as the substrate. In vitro and in vivo tests with different doses of endrin revealed a very significant pattern of enzyme inhibition. The inhibition of alkaline phosphatase activity was gradual with the addition of endrin, whereas acid phosphatase showed an initial increase in activity with a subsequent decrease after the addition of higher doses of endrin.

5.5 MAMMALS

5.5.1 Domestic

5.5.1.1 Metabolism

5.5.1.1.1 Exposure, distribution, and accumulation. Domestic herbivores are exposed to endrin primarily through ingestion of endrin-treated foliage. Inhalation of vaporized endrin and absorption through the skin following direct application of endrin to the animals represent other known routes of exposure.

Little is known of the transport and distribution of endrin in mammals. No evidence of storage in any particular tissue or organ, other than fat, has been found. However, residues of endrin ranging from 0.001 to 23.7 ppm have been detected in a variety of mammalian tissues.

Studies by Brooks (1969) demonstrated that steers, lambs, and hogs receiving 0.1 ppm endrin in the diet for 12 weeks showed little tendency to deposit endrin in body tissues. Continuous feeding of endrin at levels up to 2 ppm resulted in a maximum body fat content of 1 ppm. Cattle with only ambient environmental endrin exposure were analyzed for the presence of endrin residues in their tissues by the U.S. Department of Agriculture in 1967. Of the 2785 animals studied, 2783 contained no endrin residues; one animal contained between 0.01 and 0.1 ppm endrin in fat and another contained between 0.11 and 0.5 ppm, also in fat. By 1971, however, similar testing revealed that endrin incidence in tissues was increasing. Of 2403 cattle tested, 42 had levels of 0.01 to 0.1 ppm endrin in fat (Spaulding, 1972).

Long et al. (1961) report high levels of storage in the adipose tissue of lambs. Higher levels were detected in the internal fat surrounding the stomach and thoracid cavity than in external fat deposits. Lambs were allowed to graze for 55 days on pastures treated with 2% endrin granules, applied six times in May and June at a rate of 0.5 lb endrin per acre. The lambs were then transferred to untreated pasture, and endrin in fat was measured after 0, 14, and 42 days. After 0 days of grazing in untreated pastures, lambs had 18.3 to 23.4 ppm endrin in internal fat and 11.5 to 14.0 ppm in external fat. After 14 days, endrin levels were surprisingly higher, with 20.3 to 23.7 ppm detected in internal fat and 14.6 to 20.1 ppm in external fat. Some loss did occur after 42 days. Internal fat levels dropped to 8.9 to 13.8 ppm, and external fat contained only 6.4 to 11.0 ppm. These findings seem to contradict later reports of Brooks (1969 and 1974b) of no storage and no retention. However, the Long et al. (1961) study did feed much higher levels of endrin to younger animals, and younger animals have more fat than adults.

Sharma and Gautam (1971) detected endrin residues in the brain and liver tissue of calves. In the domestic dog, endrin was reportedly detected in the abdominal viscera (Reins et al., 1966) as well as in fat (Richardson et al., 1967). The latter authors fed endrin to dogs at a rate of 0.1 mg/kg per day for 128 days. Endrin did not accumulate in the following tissues: blood, heart, liver, kidney, pancreas, spleen, lung, or muscle. However, endrin had accumulated in fat to levels of 0.76 ppm.

5.5.1.1.2 Transformation and elimination. Biotransformation of endrin in mammals is discussed in detail in Sect. 6.2.3.

With the exception of endosulfan, endrin is the least persistent in mammals of all the commonly used pesticides. Endrin is rapidly metabolized and eliminated from vertebrate tissues (Brooks, 1974b). Excretion occurs through the milk as well as through the urine and feces. Residue levels in excess of 0.25 ppm on a fat basis were detected in the milk of 40 Wisconsin dairy herds between 1964 and 1967 (Moubry et al., 1968a). Endrin was presumably retained in the milk fat for up to four weeks. However, the quantities of endrin ingested during that period were not controlled. Extracted milk fat from one of these herds average 6.76 ppm endrin. One week following the removal of endrin sources, the average endrin levels decreased to 0.81 ppm. After four weeks the endrin levels in extracted milk fat had decreased to 0.13 ppm.

Williams and Mills (1964) studied the appearance of endrin in cow's milk under controlled feeding conditions. Cows were fed for 35 days with a combination feed supplemented with 0.05, 0.15, or 0.30 ppm endrin. Endrin concentratins in the milk increased progressively at all three exposure levels during the first few days of feeding. When 0.15 or 0.3 ppm endrin was ingested, the concentration in milk reached a maximum at approximately the 15th day. At an exposure level of 0.05 ppm, the plateau level in milk was not attained until the 13th day. When ingestion of endrin ceased, residues in milk declined sharply. Following 20 days on an endrin-free diet, detectable (greater than 0.001 ppm) levels were present only in milk samples from cows fed the highest levels of endrin (0.3 ppm). It is possible that these data were not representative for endrin alone, since several pesticides were fed together. The rate of transfer into milk might have been affected by other pesticides in the mixture.

Endrin is apparently excreted in milk in higher concentrations when fed as a residue on hay than when fed dissolved in soybean oil (Ely et al., 1957). When endrin-treated hay was fed to cows in doses of 0.05 mg endrin/kg, no measurable amounts were detected in milk. Somewhat larger doses (0.06 to 0.11 mg/kg) resulted in concentrations of 0.1 to 0.2 ppm. Ingestion of 0.11 to 0.24 mg/kg endrin in soybean oil, on the other hand, was insufficient to produce detectable residues in milk. However, doses ten times as large (1.11 to 2.33 mg/kg) did result in 0.15 to 0.27 ppm endrin in milk on a fat basis. In general, a total daily endrin intake of greater than 20 mg as a residue sprayed in forage is necessary for excretion of measurable quantities of endrin in milk.

In another study (Saha, 1969) the average concentration of endrin excreted in milk was found to be 0.05 to 0.08 ppm. The ratio of residue in milk to feed was 0.07. This rate of excretion was higher than that for DDT, heptochlor, teraphene, chlordane, and methoxychlor.

The levels of endrin in milk following ingestion of feed containing various concentrations of endrin are presented in Table 5.14.

Table 5.14. Endrin residues in cow milk after feeding

Feed (ppm)	Milk (ppm)	Feeding (days)	Source
0.1	0.01	84	1
0.25	0.02	84	
0.75	0.04	84	
2.00	0.10	84	
0.05	0.004	35	2
0.15	0.010	35	
0.30	0.018	35	
1.9	<0.05	48–63	3
2.8	0.14	48–63	
3.7	0.15	48–63	

Sources: 1 — Saha, 1969;
2 — Williams and Mills, 1964;
3 — Ely et al., 1957.

5.5.1.2 Effects

5.5.1.2.1 Acute exposure. Endrin is highly toxic to mammals in large doses. Adverse effects have been reported following oral, respiratory, intravenous, and dermal exposure. Death from endrin has been reported for the following domestic mammals: horse, rabbit, goat, dog, cat, cow, and hamster. The dose lethal to 50% of the test animals is presented in Table 5.15. Female mammals appear more susceptible than males to endrin poisoning (Ely et al., 1957), possibly because females store more fat. For this reason, horse LD_{50}s are given as 10 mg/kg for mares and 35 mg/kg for stallions and geldings (Catcott and Smithcors, 1972). Other LD_{50}s ranged from a low of 2.5 mg/kg for dogs to a high of 50 mg/kg for goats.

There are no known reports of dogs dying from environmental exposure to endrin. However, the lethality of endrin to dogs has been demonstrated through laboratory studies. Reins et al. (1966) injected up to 25 mg endrin per ml intravenously into dogs. The increased blood pressure and heart rate that resulted led to convulsions and finally death. At an injection level of 2 mg/kg, 40% of the dogs died. At a rate of 3 mg/kg, 75% died. Between 5 and 10 mg/kg there was 100% mortality (Reins et al., 1966). Brooks (1974b) reported mortality in dogs from 25 ppm endrin in diets.

Table 5.15. Endrin lethal toxicity to mammals

Domestic animals	LD_{50} (mg/kg)	Source
Horse	10–35	1
Hamster	10	8
Rabbit	5–10	2–7
Goat	25–50	2–4
Dog	2.5	3
Wild animals		
Big brown bat	5–8	5
Pine mouse	2.6	6

Sources: 1 — Catcott and Smithcors, 1972;
2 — Pimentel, 1971;
3 — Reins et al., 1966;
4 — Tucker and Crabtree, 1970;
5 — Luckens and Davis, 1965;
6 — Petrella and Webb 1973;
7 — Negherbon, 1959;
8 — Ottolenghi et al., 1974.

Sharma and Gautam (1971) report cows dying from both ingestion and inhalation of endrin. Hematological changes and lesions were reported.

Cattle mortality following dermal exposure has been reported by Sharma and Gautam (1971), Moubry et al. (1968a), and Jerome (1958). The last author reported the most extreme example of accidental poisoning. After being sprayed for flies with a 0.34% endrin solution, 218 out of 370 cattle were affected; the mortality rate was 71%. Acute symptoms were: tetanic seizure, spasmodic actions, grinding teeth, salivation, and falling. Peracute symptoms were: twitching, blinking, and jumping, as well as respiratory and cardiac arrest. Postmortem examinations revealed gross lesions, myocardial hemorrhages, tissue anoxia, and cyanosis.

Sharma and Gautam (1971) reported both dermal and oral poisoning in calves. An endrin solution as small as 0.04% sprayed on calves caused death within 6 hr. Oral doses of 4.5 to 5.5 mg per pound of body weight were also lethal.

Goats appear to be more resistant to lethal doses of endrin than do other domestic mammals. LD_{50} varies from 25 to 50 mg/kg endrin. Acute symtoms of poisoning were ataxia, slowness, tremors, tracheal congestion, prostration, and convulsions. Goats died from one to five days after oral treatment (Tucker and Crabtree, 1970).

5.5.1.2.2 Chronic exposure. Chronic exposure to endrin adversely affects physiology, behavior, and reproduction; the last two are least documented in domestic animals. Sublethal physiological effects observed in some species are presented in Table 5.16. The symptoms of endrin poisoning range from subacute nervous spasms and slight cyanosis (Jerome, 1958) to lesions, convulsions, and increased blood pressure and heart rate.

Table 5.16. Endrin toxicity in domestic mammals — sublethal

Mammal	Storage in tissue	Effects (physiological)	Source
Cat	Unknown	Hematological changes and lesions	1
Calf	Brain and liver	Hematological changes and lesions	1
Cow	Fat and milk	Convulsions, high breathing rate, lesions, tissue anoxia	2
Dog	Abdominal viscera and fat	Changes in blood pressure and heart rate, convulsions	3, 6
Rabbit	Unknown	Body and organ weight increased	4
Goat	Unknown	Ataxia, tremors, convulsions	5

Sources: 1 — Sharma and Gautam, 1971;
2 — Jerome, 1958;
3 — Reins, et al., 1966;
4 — Takahama et al., 1972;
5 — Tucker and Crabtree, 1970;
6 — Richardson et al., 1967.

Singh et al. (1977) treated buffalo calves with a 20% emulsible concentrate formulation of endrin orally at a rate of 7.5 mg/kg body weight. Within 2 to 3.5 hr after treatment the calves went into convulsions lasting approximately 5 min with a significant ($P < 0.01$) rise in the level of blood glucose. Cholinesterase activity of the erythrocytes was gradually restored as the signs subsided and returned to nearly normal on the sixth day after treatment.

The dose which can initiate the onset of symptoms of endrin poisoning in calves is 2.0 mg/lb body weight (Sharma and Gautam, 1971). The symptoms in calves and the results of postmortem analysis were as follows:

- nervous excitement,
- convulsions,
- accelerated respiratory rate,
- depressed erythrocyte count,
- increased total leukocyte count (lymphocytes),
- lesions of the gall bladder and kidney,
- albumin in the urine,
- congested kidney and lungs,
- endrin-positive brain and liver.

Long et al. (1961) reported that lambs grazing on endrin-treated pastures stored up to 23.7 ppm endrin in fat without exhibiting any symptoms of endrin poisoning. In fact, there was not even a decrease in weight gain.

Dogs fed 0.1 mg/kg per day had blood levels of endrin of less than 0.001 ppm and showed no toxicity symptoms (Richardson et al., 1967). Brooks (1974b) reported no effect in dogs fed 1 ppm endrin. However, a diet of 5 ppm/day increased liver and kidney weights. No evidence of endrin-related carcinogenesis has been reported (Brooks, 1974b).

Small doses of endrin may result in symptoms which might go unnoticed outside of the laboratory or be improperly diagnosed. For example, cows may show lack of appetite or decreased milk production (Ely et al., 1957). In some animals, reproduction rates may be lowered (Pimentel, 1971; Ottolenghi et al., 1974). Ingestion of small amounts of endrin (0.1 mg/kg) by domestic rabbits resulted in increased weights of liver, kidney, heart, lung, and whole body as compared with controls. Such effects would not be obvious under natural conditions.

Not enough long-term or consistent data are available on the sublethal effects of endrin, and endrin toxicology is not well known to veterinarians.

5.5.2 Wild Mammals

5.5.2.1 Exposure and metabolism. Wild mammals are exposed to endrin following application of the pesticide to crops, orchards, and pastures. Uptake may occur from dermal absorption, oral ingestion of contaminated insects and vegetation, and by grooming the fur (Morris, 1970). Presumably, wild mammals inhale endrin, as do domestic mammals.

While extensive information is available on the effects of endrin in several species of wild mice, very little has been published for other species. Most of the literature deals with herbivores and insectivores, with little available data concerning endrin in carnivores and omnivores.

Traces of endrin have been found in fat as well as other tissues of wild mammals. Trace amounts of endrin (0.001 to 2.8 ppm) have been reported in the fat of white-tailed deer (*Odocoileus virginianus*) (Cotton and Herring, 1971), pronghorn antelope (*Antilocapra americana*) (Moore et al., 1968), and mule deer (*O. hemionus*) (Stickel, 1973; Jewel, 1966). Wilson (1966) reported that the blubber of porpoises (Phocaenidae) contained 0.05 to 2.0 ppm endrin. Jewell (1966) reported that deer muscle tissue also had traces of endrin (0.072 ppm). Ferrel (1963) reported that rabbits (*Lepus sp.* and *Sylvilagus sp.*) had endrin residues of 1.6 ppm in kidney and liver tissues. Hooven (1957) reported residues of endrin in the following tissues: the stomach and intestine of the grey squirrel (*Sciurus carolinensis*) (1.17 and 1.55 ppm respectively) and the intestine of chickaree (*Tamiasciurus douglasi*) (0.56 ppm).

These data do not necessarily indicate endrin storage. However, Morris (1968) hypothesized that endrin was stored in fat and translocated to the nervous system during times of stress. Webb et al. (1972), on the other hand, report that endrin was not stored in bodies of wild mice (*Pitymys pinetorum*) in detectable amounts.

Hepatic microsomes from mice converted endrin to the metabolite 12-OH-endrin (Petrella et al., 1974). Oral dosing of mice with endrin resulted in the production of one fecal and three urinary metabolites. The endrin metabolites were found to be excreted in the following proportions: resistant mice, 18% in urine and 53% in feces; susceptible mice, 23% in urine and only 27% in feces. The greater amount of metabolites released by resistant mice (71%) than by susceptible mice (50%) is well documented (Webb et al., 1972, 1973; Petrella and Webb, 1973; Petrella et al., 1974; Petrella et al., 1977). Thus, resistance may be due, in part, to endrin degradation and the subsequent excretion of the metabolites.

5.5.2.2 Effects. VonRumker et al. (1974) defined wild-animal toxicity in terms of population losses in habitats. "Highly toxic" means severe losses occur if the pesticide is used over a given habitat. "Moderately toxic" means moderate losses. "Slightly toxic" means slight loss or injury to nontarget species. "Relatively nontoxic" was defined as no loss or injury. Endrin seems to display all these levels of toxicity.

5.5.2.2.1 Acute toxicity. Endrin (2.0 lb/acre, or 2.2 kg/ha) has been shown to be highly toxic to mouse populations for periods of up to 70 days (Dana and Shaw, 1958). Mortality in mouse populations of 33 to 100% was observed following field applications of endrin (0.5 to 2.5 lb/acre, or 0.55 to 2.75 kg/ha). Table 5.17 lists mouse species affected at varying rates of field spraying.

In addition to mice, the following species of mammalian wildlife have been reported as receiving lethal doses of endrin: big brown bat (*Eptesicus fuscus*), grey squirrel (*Sciurus carolinensis*), chipmunk (*Tamais striatus*), cottontail rabbit (*Sylvilagus sp.*), jackrabbit (*Lapus sp.*), woodchuck (*Marmota monax*), and shrew (*Sorex sp.*). The only species with established endrin LD_{50}s are pine mouse, 2.6 mg/kg (Petrella and Webb, 1973); brown bat, 5 to 8 mg/kg (Luckens and Davis, 1965); and rabbit, 5 to 7 mg/kg (Negherbon, 1959).

Wildlife symptoms of acute endrin toxicity are described for grey squirrels and chipmunks by Hamrick (1969); they are tremors and reduced vitality. Cotton rats exhibited severe tremors (Hamrick, 1969). Luckens and Davis (1965) described tremors in bats before death by endrin poisoning. Schindler (1956) described abnormal movements in wild mice for several days preceding 100% mortality in a treated field.

Table 5.17. Applications of endrin in field to kill mice

Species	Rate of Application (lb/acre)	Source
Pitymys pinetorum	2.5	1
Microtus sp.	2.0	2, 5
	1-1.5	3
	1.6–2.4	4
	0.5	6
Peromyscus maniculatus	0.5	6

Sources: 1 — Horsfall and Webb, 1966;
2 — Dana and Shaw, 1958;
3 — Schindler, 1956;
4 — Wolfe, 1957;
5 — Snyder, 1963;
6 — Morris, 1970.

5.5.2.2.2 Chronic toxicity. Endrin has been shown to have subacute effects on wildlife populations by affecting reproduction, behavior, ecology, and possibly evolution. The last can only be verified through long-term observations of adaptation to endrin.

Sublethal effects of endrin are summarized in Table 5.18. Morris (1972) describes sublethal effects of endrin due to direct toxicity as either behavioral disorders or delayed reproduction. Indirect toxicity may result when endrin is combined with environmental stress, such as cold. Figure 5.6 shows various pathways of direct and indirect biocide stress on small-mammal populations.

Reproductive disorders can occur in wild mammals due to endrin exposure. Reproductive rates have both increased and decreased due to endrin. Increases are due to larger amounts of territory (food, water, and cover) available to survivors after population reduction due to endrin. For example, continuing studies by Morris (1968, 1970, 1971a, b, 1972) have demonstrated that significant reductions occur in *Microtus* populations after endrin spraying. However, populations recovered quickly, and in two to three years demonstrated higher densities than control groups. Morris (1972) attributes this phenomenon to repopulation and higher survival rates of the young of surviving mice. The remaining low-density *Microtus* population was able to expand territories and avail itself of more food and cover. Reproductive potential was unharmed. Driggers' (1971) hypothetical conclusion was that endrin killed mouse predators and parasites, allowing the population to prosper.

Table 5.18. Endrin toxicity on wild mammals -- sublethal

Mammal	Classification	Storage in Tissue	Effects	Source
Deer mouse	*Peromyscus sp.*	Fat	Starvation stress, unstable population, and reproduction	1 2, 3
Meadow mouse	*Microtus sp.*		Reproduction altered	4
Big brown bat	*Eptesicus fuscus*		Tremors	5
Gray squirrel	*Sciurus carolinensis*		Tremors	6
Cotton rat	*Sigmodon hispidus*		Tremors	6
White-tailed deer	*Odocoileus virginianus*	Fat	None known	7
Mule deer	*O. hemionus*	Fat, muscle	None known	8
Porpoises	*Phocaenidae*	Blubber	None known	9

Sources: 1 — Morris, 1968;
2 — Morris, 1970;
3 — Morris, 1971b;
4 — Snyder, 1963;
5 — Luckens and Davis, 1965;
6 — Hamrick, 1969;
7 — Cotton and Herring, 1971;
8 — Jewell, 1966;
9 — Wilson, 1966.

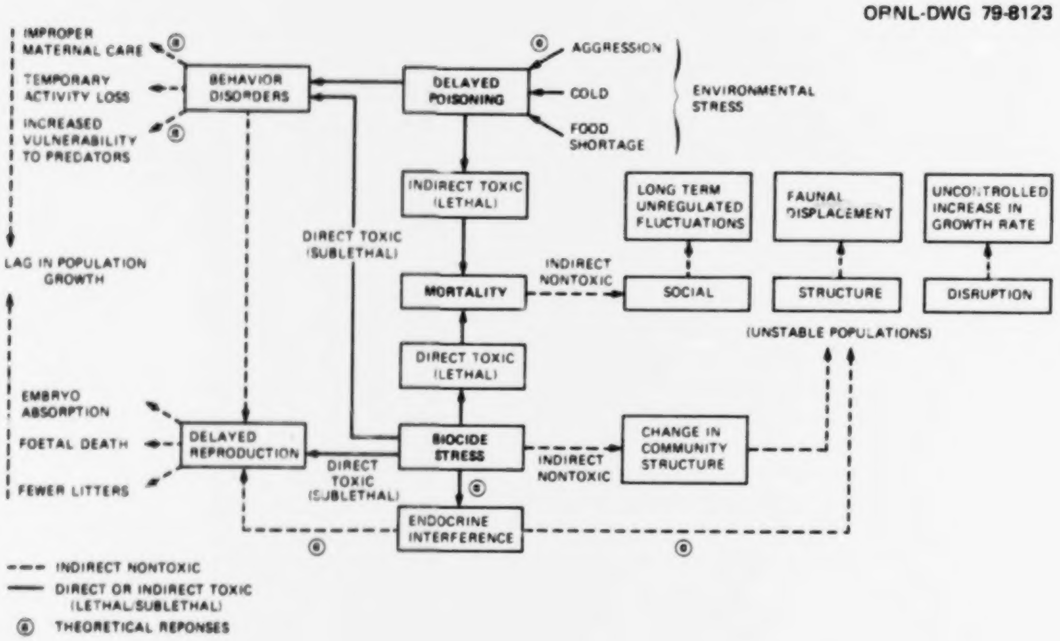
ORNL-DWG 79-8123
IMPROPER MATERNAL CARE
TEMPORARY ACTIVITY LOSS
INCREASED VULNERABILITY TO PREDATORS
BEHAVIOR DISORDERS
DELAYED POISONING
AGGRESSION
COLD
FOOD SHORTAGE
ENVIRONMENTAL STRESS
INDIRECT TOXIC (LETHAL)
LONG TERM UNREGULATED FLUCTUATIONS
FAUNAL DISPLACEMENT
UNCONTROLLED INCREASE IN GROWTH RATE
DIRECT TOXIC (SUBLETHAL)
LAG IN POPULATION GROWTH
MORTALITY
INDIRECT NONTOXIC
SOCIAL
STRUCTURE
DISRUPTION
(UNSTABLE POPULATIONS)
DIRECT TOXIC (LETHAL)
EMBRYO ABSORPTION
FOETAL DEATH
FEWER LITTERS
DELAYED REPRODUCTION
BIOCIDE STRESS
DIRECT TOXIC (SUBLETHAL)
INDIRECT NONTOXIC
CHANGE IN COMMUNITY STRUCTURE
ENDOCRINE INTERFERENCE
INDIRECT NONTOXIC
DIRECT OR INDIRECT TOXIC (LETHAL/SUBLETHAL)
THEORETICAL REPONSES

Fig. 5.6. Generalized model of the ways in which a biocide may affect a small-mammal population. Source: R. D. Morris, *Can. J. Zool.* 50(6): 885-896 (1972). Copyright 1972, National Research Council of Canada.

Peromycus populations, on the other hand, did not fare as well in the same experiment. In 1970, Morris concluded that *Peromycus* populations were severely reduced by endrin spraying and did not recover after a few years. *Peromycus* showed long-term toxicological effects. No new juveniles were found after spraying with endrin. However, in 1972, Morris found that *Peromycus* were actually escaping from his enclosure. His earlier findings could have been affected by this.

Other authors have found reduced reproductive potential due to endrin. Good and Ware (1969) and Snyder (1963) showed reduction in number of litters of *Microtus* after endrin application of 0.6 to 2.0 lb/acre. Snyder (1963) fed endrin to *Microtus* and found lower reproductive rates due to reduced corpora lutea, implantation, and viable embryos. Ottolenghi et al. (1974) fed endrin to mice during gestation and found postnatal mortality of unweaned juveniles. This could have been due to either endrin's direct effects on fetuses or due to the presence of endrin in milk.

Differential survival rates of wild mammals can have long-term ecological or evolutionary effects which are still unknown. Morris' studies demonstrated the potential for *Peromyscus* populations to be replaced by *Microtus* in areas where endrin spraying is continuous year after year.

Pine mice (*Pitymys pinetorum*), on the other hand, have been shown to develop an inheritable resistance to endrin. Webb et al. (1973) report that pine mice from areas sprayed regularly with endrin had an LD_{50} as high as 36.42 mg/kg. Pine mice from unsprayed areas had an LD_{50} as low as 1.37 mg/kg. The endrin-resistant mice also exhibited a twofold cross-resistance to dieldrin. Webb and Horsfall (1967) earlier found pine mice with a 12-fold greater tolerance to endrin than susceptible mice from unsprayed fields.

The resistance to endrin can be lost over time if endrin treatment is stopped. Webb et al. (1973) report that three years after the last endrin application the LD_{50} in pine mice decreased from 36.42 mg/kg to 18.91 mg/kg. Webb et al. (1973) demonstrated that endrin resistance might be hereditary as well as acquired. When susceptible males were crossed with susceptible females, LD_{50} values of progeny averaged 4.97 mg/kg. When susceptible and resistance mice were crossed with resistant, LD_{50}s for progeny were 21.08 mg/kg. Therefore, in a field sprayed regularly with endrin, resistant mammals would survive and produce resistant young. At the same time, susceptible species would be killed or reproductive rate affected. The long-term effect would be a different population structure, loss of some species, and domination by other species.

Morris (1972) also discusses potential sublethal behavior changes in wild mammals. These are related to maternal care, activity, and vulnerability to predators. Endrin could also disrupt social patterns in populations, particularly when there is a sudden "vacuum" created by mortality. Decreased competition leads to expansion of territories and changes in rates of immigration and emigration, resulting in unstable and fluctuating populations of small mammals. Predation rates may also increase (Hamrick, 1969), secondarily affecting larger predator populations as well.

Stickel (1973) reports that predatory mammals accumulate the highest residues of chlorinated hydrocarbons. No data are given for endrin. Schneeweis et al. (1974) report that endrin residues were below 1 ppm in tongue and muscle tissue of timber wolves (*Canis lupus*) from Canada. These data are inconclusive because wolves were not feeding in endrin-sprayed areas; no fat was sampled, and minimum standards used were those established for birds. No other mammalian predator data for endrin are known to date. More research is necessary to determine whether endrin is accumulated in carnivores, which feed on prey with high levels of pesticides. No studies have been conducted on predators feeding on resistant populations of mice.

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6. BIOLOGICAL ASPECTS IN HUMANS

6.1 SUMMARY

Endrin, the most acutely toxic of all the cyclodiene insecticides, is highly hazardous to all mammals regardless of route of exposure. Endrin is less persistent, however, than most other organochlorine compounds. It is quickly metabolized in mammals and excreted as hydrophilic metabolites. Endrin is eliminated much more rapidly than DDT or dieldrin from mammalian tissues.

Human exposure to endrin occurs through the diet, through inhalation, and through dermal contact. The average dietary intake in the United States from 1965 to 1970 was 0.005 μg/kg body weight per day. This level is far below the maximum acceptable daily intake of 0.002 mg/kg body weight established by the World Health Organization. Respiratory or dermal exposure to endrin is possible during manufacture and distribution but is more likely to result, directly or indirectly, from agricultural uses.

Outbreaks of human poisoning have resulted from accidental contamination of foods and have been traced to doses as low as 0.2 mg/kg. The symptoms usually observed in victims of endrin poisoning were convulsions, vomiting, abdominal pain, nausea, dizziness, and headache. In severe cases, convulsive seizures of several minutes duration may be followed by semiconsciousness. Convulsions can result in death through respiratory failure. The toxicity of endrin appears to result primarily from the effect of endrin on the central nervous system. Endrin loads in the general population, however, are probably not sufficient to induce changes in brain function.

Endrin poisoning can be fatal. The mean lethal dose (LD_{50}) values for acute oral exposures of laboratory animals range from 3 to 43 mg/kg body weight.

Chronic exposures to low levels of endrin can be toxic to mammalian species. Significant mortality has been observed in mice exposed to 2 ppm endrin in the diet for seven months. Fatalities were observed following inhalation exposure of animals to 0.36 ppm endrin vapor for 7 hr on each of 130 days.

There is no evidence indicating carcinogenicity of endrin. Endrin can, however, cause mutations in mammalian cells. Endrin is teratogenic in mice and hamsters. Exposure to endrin causes an increase in fetal and postnatal mortality, but fertility of parents is generally not affected.

6.2 METABOLISM

6.2.1 Exposure

The general population is exposed to very little endrin in the diet. In the United States, Duggan and Lipscomb (1969) and Duggan and Corneliussen (1972) found that the total average intake from food ranged from about 0.01 μg/kg body weight per day in 1965 and in 1968, to 0.0005 μg/kg body weight per day in 1970. The six-year average intake was 0.005 μg/kg body weight per day. These levels are far below the maximum acceptable daily intake of 0.002 mg/kg body weight which has been established by the Joint FAO/WHO Meeting on Pesticide Residues in Food (World Health Organization, 1973).

Agricultural workers, anyone using endrin in a home garden, and those involved in manufacture or distribution of endrin might be exposed to endrin through inhalation or dermal contact. Probably the greatest hazard associated with the use of endrin occurs when measuring and pouring the emulsifiable concentrate material (Wolfe et al., 1963).

The most significant occupational exposure to endrin comes during spraying of fields. Kale and Dangwal (1970) measured endrin concentrations during cropland spraying ranging from trace amounts to 7.0 $\mu g/m^3$. In dust or spray machine operations, dermal exposure is almost always greater than respiratory exposure (Wolfe et al., 1963). Dermal exposure during orchard spraying is likely to range up to 3 mg/hr, while respiratory exposure would be likely to reach 0.01 mg/hr (Wolfe et al., 1963; Wolfe et al., 1967).

A threshold limit value of 0.1 $\mu g/m^3$ for an 8-hr time weighted average occupational exposure has been established by OSHA (U.S. Code of Federal Regulations, 1972).

6.2.2 Distribution and Accumulation

Humans do not tend to store endrin in significant quantities. No residues were detected in plasma, adipose tissue, or urine of workers occupationally exposed to endrin (Hayes and Curley, 1968). Measurable levels of endrin have not been found in human subcutaneous fat or blood, even in areas where it is extensively used, as in India or the lower Mississippi area (Brooks, 1974b). Despite its high acute toxicity (Sect. 6.3.2), endrin is a relatively nonpersistent pesticide in man. Endrin residues are only detected in body tissues of humans immediately after an acute exposure.

As a result of acute endrin poisoning, high endrin content has been observed in blood and urine, but no endrin has been detected in cerebral spinal fluid (Cobel et al., 1967). Poisoned humans have been reported to have an endrin content as high as 400 ppm in fat and 10 ppm in other tissues (Cobel et al., 1967). The 400-ppm value was obtained by a bioassay technique no longer regarded as dependable. The 10-ppm value was determined in a patient who died after repeated exposures to endrin. Much lower levels of endrin were found at autopsy in body tissues of endrin poisoning victims during an outbreak in Saudi Arabia (Table 6.1). Samples of blood and urine taken from patients 29 to 31 days after the outbreak (Sect. 6.3.1.1) were uniformly negative for endrin (Curley et al., 1970). Low blood levels were found in three humans who recovered after accidental ingestion of endrin. In one case the concentration of endrin in the blood 30 min after convulsions occurred was 0.053 ppm, and 20 hr later it was 0.038 ppm. The same patient excreted 0.02 ppm endrin in his urine during the next 24 hr (Cobel et al., 1967). Endrin is not found in blood, urine, or tissue samples from the general population or even from workers who manufacture and formulate endrin (Hayes and Curley, 1968).

Table 6.1. Endrin concentrations found in victims of endrin poisoning in Saudi Arabia

Sample	Endrin concentration (ppm)
Blood	0.007-0.032
Urine	0.004-0.007
Vomitas	5.24
Tissues (autopsy) from:	
Stomach wall	0.16
Liver	0.685
Kidney	0.116

Source: A. Curley, R. W. Jennings, H. T. Mann, and V. Sedlak, *Bull. Environ. Contam. Toxicol.* 5(1): 24-29 (1970). Copyright 1970 Springer-Verlag.

Oral administration of ^{14}C-labeled endrin to rats in daily doses of 8 μg in peanut oil for 12 days resulted in equilibrium after six days. After 12 days the spleen had the highest concentration, 3 ppm; the blood, 1.1 ppm; the skin and subcutaneous adipose tissue, 0.74 ppm; the peritoneal adipose tissue, 0.38 ppm; and the brain, 0.25 ppm. Four days after cessation of feeding, male rats were found to have retained only 5.3% of the total radioactive material given, and the females, 15% (Klein et al., 1968a).

Richardson et al. (1967) fed endrin to nine-month-old dogs for 128 days at a level of 0.1 mg/kg body weight per day. Blood concentrations during the experiment ranged from 0.002 to 0.008 ppm. At the end of the experiment, concentrations in adipose tissue ranged from 0.3 to 0.8 ppm; heart, pancreas, and muscle were at the lower end of this range, while the concentration in liver was 0.077 to 0.084 ppm; kidneys and lungs had similar concentrations.

The amounts of endrin found in the tissues of dogs that survived after being fed for almost six months on diets containing endrin in concentrations of 4 to 8 ppm were: 1 ppm in fat; 1 ppm in liver; and 0.5 ppm in kidneys (Treon et al., 1955).

6.2.3 Biotransformation and Elimination

Endrin is metabolized and excreted more rapidly than other chlorinated hydrocarbon insecticides (Jager, 1970). There is good evidence that endrin is quickly metabolized in mammals and excreted as hydrophilic metabolites.

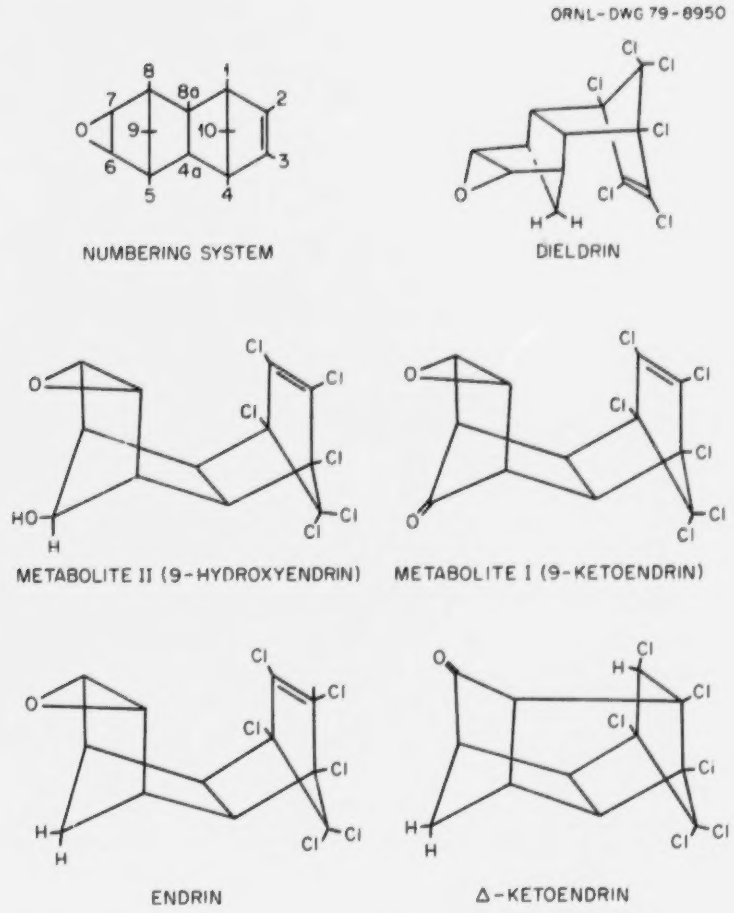
Experiments of Korte (1967) demonstrate that conversion of endrin to metabolites takes place in the mammalian organism itself and is not caused by microorganisms in the intestinal tract. The liver appears to be the site of biotransformation (Korte et al., 1970; Jager, 1970).

In vitro studies appear to support the hepatic metabolism of endrin. A metabolite behaving as a monohydroxy derivative was produced when endrin was incubated at 30°C for several hours with both rat liver and pig liver microsomes and NADPH (Brooks, 1969). Formation of the derivative was suppressed by sesamex, an inhibitor of microsomal oxidations.

Endrin is apparently degraded to derivatives which are less toxic to mammalian organisms. When ^{14}C-labeled endrin was given orally and intravenously to rats, the ketoanalogue of endrin and other hydrophilic metabolites were present in trace amounts in the urine (Klein et al., 1968a). Since the acute oral LD_{50} of ketoendrin to rats (62 mg/kg) is higher than that of endrin (25 mg/kg), the rearrangement would be detoxification reaction (Brooks, 1969).

Endrin is metabolized in the rat to at least three metabolites (Baldwin et al., 1970). One is 9-ketoendrin, which is found in the urine. The other two metabolites are excreted in the feces and have not been found in body tissues (Baldwin et al., 1970). In all experiments utilizing rats, an isomer of 9-hydroxyendrin accounts for 95% of the radioactivity excreted (Korte et al., 1970). This isomer has the hydroxyl group endo with respect to the epoxy group (Fig. 6.1). The third metabolite is a monohydroxylated endrin, but the hydroxyl group is not on carbon 9 (Baldwin et al., 1970). Methanobridge hydroxylation occurs in the rat, and the corresponding ketone is found in adipose tissue, liver, and brain.

Rabbits excreted radioactivity after intravenous administration of ^{14}C-labeled endrin mainly in the urine and only as metabolites (Korte et al., 1970). Hydroxylation seems to occur in the rabbit without major skeletal rearrangement (Brooks, 1974b). Four metabolites were found in order of decreasing polarity in the ratio of 1:1:3:1. The second most polar one is identical with the isomer of 9-hydroxyendrin, which was the main metabolite found in rats. When NADH was added to 100 g of rabbit liver homogenate, 38 μg of endrin was metabolized within 72 hr to an extent of 39.6% (Korte et al., 1970).



NUMBERING SYSTEM

DIELDRIN

METABOLITE II (9-HYDROXYENDRIN)

METABOLITE I (9-KETOENDRIN)

ENDRIN

Δ-KETOENDRIN

Fig. 6.1. Structures of the metabolities I and II, endrin, ketoendrin, and dieldrin. Source: Reprinted with permission from M. K. Baldwin, J. Robinson, and D. V. Parke, *J. Agric. Food Chem.* 18(6): 1117-1123 (1970). Copyright 1970 American Chemical Society.

Oxidations without skeletal rearrangement predominate in mammals. There is, as yet, no evidence for epoxide ring hydration, and *trans*-diol formation may be sterically difficult (Brooks, 1974b).

Studies indicate attainment of plateau storage levels for endrin. For a given level of endrin intake, a steady state should be achieved in which the rate of intake is balanced by the rate of elimination (Brooks, 1969). Rats given a daily oral dose of 8 μg ^{14}C-labeled endrin for 12 days reached a plateau level of endrin storage in nine to ten days (Brooks, 1969). When endrin is given orally to rats at a daily dose corresponding to 0.4 ppm in the diet, a steady state of storage is reached after about six days for male and female rats (Korte, 1967; Korte et al., 1970). The storage level for females (27%) is about twice as high as that for males (14%). Four days after administration was stopped, males retained only 5.3% and females 15% of the administered radioactive substance.

At high dosage levels, excretion of endrin appears to be slower. Korte et al. (1970) determined the biological half-life of endrin to be one to two days after an oral dose of 16 or 64 μg/kg body weight. However, after an oral dose of 128 μg/kg body weight, the half-life increased to approximately six days.

Females metabolize endrin more slowly than males (Brooks, 1969). The biological half-life of endrin in male rats receiving 0.4 ppm in the diet is two to three days; in females approximately four days (Korte et al., 1970). Following intravenous injection of 200 μg of ^{14}C-labeled endrin per kilogram body weight in two doses, male rats retained 5.2% and females 12.1% of the administered dose after 24 hr. The radioactivity was totally excreted as metabolites (Korte et al., 1970). Similar results were obtained by Jager (1970) in feeding experiments. When ^{14}C-labeled endrin was fed to male and female rats, the males excreted 60% of it in the feces within the first 24 hr, and the females only 39%; less than 1% was excreted in the urine. Of the total radioactivity excreted in the feces, 70 to 75% occurred in the form of hydrophilic metabolites; the remainder was in unchanged endrin. Twenty-four hours after the last dose, only hydrophilic metabolites were excreted (Jager, 1970).

The difference in rates of metabolic conversion between endrin and dieldrin is likely to be related to the stereochemistry of the endrin molecule, which predisposes it to skeletal rearrangements. When dogs, fed a daily diet containing 0.1 mg/kg dieldrin, were compared with others similarly fed with endrin, the blood dieldrin level approached a maximum after 114 to 121 days, but there was no accumulation of endrin in the blood (Richardson et al., 1967).

Body content of endrin declines fairly rapidly after a single dose or when a continuous feeding experiment is terminated. When exposure ceases, endrin concentration in tissues declines, and the rate of decline is proportional to tissue concentration at that time (Brooks, 1969).

Most endrin is excreted in the feces, much of it through the bile. The liver has been identified as the major excretory organ for endrin in the rat. The amount of radioactivity in the feces of rats, administered ^{14}C-labeled endrin intravenously, reflects the amount of radioactivity entering the gastrointestinal tract via the bile (Cole et al., 1970).

Cole et al. (1970) studied rates of excretion of radiocarbon-labeled endrin in whole rats, bile-fistula rats, and isolated perfused rat livers. Over 90% of the excreted radioactivity was found in the feces of the intact animals and in the bile of fistula animals. Fifty percent of the radioactive endrin was excreted within the first day. In fistula animals, 50% of the endrin radioactivity was excreted in the bile in approximately 1 hr in the perfusion experiments (Cole et al., 1970). A similar set of experiments using radiolabeled dieldrin indicated that excretion of radioactivity from endrin was greater than that from dieldrin in the feces of intact animals, bile of bile-fistula animals, and bile of isolated, perfused rat liver preparations.

The liver appears to be the major controlling factor in the different rates of excretion of radioactivity from ^{14}C-labeled endrin and ^{14}C-labeled dieldrin. The more rapid fecal elimination of endrin can be explained by its more rapid biliary excretion (Cole et al., 1970). Since endrin is excreted more rapidly than dieldrin, it is less available for storage.

6.3 EFFECTS

6.3.1 Human Exposure

6.3.1.1 Accidental poisonings. Several incidents of human endrin poisoning have been reported in the literature.

The first report of acute endrin poisoning was from Wales in 1956. Sacks of flour were contaminated during shipment in a railway car in which 3 gal of endrin had leaked several months earlier. Bread made in a bakery from this flour resulted in almost 60 cases of acute endrin poisoning; no deaths occurred. The bread was contaminated with 150 ppm endrin (Davies and Lewis, 1956). Between 1958 and 1963, endrin poisoning in the state of Andra Pradesh, India, increased from two a year to 140 a year, paralleling increased use of the insecticide (Reddy et al., 1966). Two three-week-old babies died from endrin poisoning in Israel in 1970. The stomach contents of one baby contained 103 ppm endrin and a trace of dieldrin. The cause was presumably a contaminated recycled glass milk bottle (Tadjer and Dore, 1971).

Between June 3 and July 15, 1967, four explosive outbreaks of acute poisoning with endrin occurred in Doha in Qatar and Hofuf in Saudi Arabia (Weeks, 1967; Curley et al., 1970). The outbreaks resulted in hospitalization of 874 persons and death of 26 persons. It was estimated that another 500 to 750 persons were poisoned but not hospitalized. Flour in a lower deck of each of two ships had been contaminated by an emulsifiable concentrate of endrin carried on an upper deck of the same hold. Poisoning resulted when the flour was made into bread and eaten (Curley et al., 1970). Three cases of accidental poisoning of infants have been reported in the United States (Cobel et al., 1967).

The most common symptoms observed in these outbreaks were vomiting, convulsions, abdominal pain, nausea, dizziness, and headache. The first indication of acute poisoning was often a sudden epileptical convulsion lasting several minutes and accompanied by frothing of the mouth. Contractions were violent and, in severe cases, followed by a sudden loss of consciousness (Cobel et al., 1967; Weeks, 1967).

An endrin intake of 0.2 to 0.25 mg/kg produced convulsions in humans during the Wales outbreak in 1956 (Davies and Lewis, 1956). Doses estimated at 5 to 50 mg/kg were responsible for the endrin poisoning in Andhra Pradesh, India. Of the 290 cases from 1958 to 1963, 60 were fatal (Reddy et al., 1966). In the majority of fatal cases, individuals died within an hour or two after ingestion of endrin. Respiratory failure was the most common mode of death (Reddy et al., 1966).

In many nonfatal endrin poisonings, a rapid recovery to a normal sense of well-being usually ensued within 24 hr, particularly in patients who vomited. In a few cases, headache, lethargy, weakness, and anorexia persisted for a few weeks (Cobel et al., 1967). With more severe poisoning, convulsions were multiple or continuous and resulted in residual damage to the central nervous system from anoxia (Cobel et al., 1967).

6.3.1.2 Occupational exposure. Versteeg and Jager (1973) report, in a study done by Shell Internationale Research, that occupational exposure to endrin in a manufacturing plant (Shell Netherlands Chemical Industries at Pernis near Rotterdam) for periods up to 17 years did not result in any persistent adverse effect as far as could be demonstrated by the following parameters: miniature chest radiograph, electroencephalogram, routine urinalysis, blood analysis, general physical examination, and a blood chemistry profile which included 12 biochemical tests. Versteeg and Jager (1973) claimed that all symptoms of endrin intoxication were fully reversible within a few days.

A study of the pattern and rate of sickness absenteeism at the Shell Netherlands Chemical Industries Plant at Pernis among endrin-exposed workers and their nonexposed colleagues from other plants revealed no significant differences between the two groups (Hoogendam et al., 1965).

Analytical tests were performed on men employed in a factory where endrin, aldrin, dieldrin, a nematocide, and certain organic phosphorus compounds were manufactured. Eleven isomers or metabolites representing six insecticides were detected in plasma samples; seven isomers or metabolites representing five insecticides were found in fat samples in the urine; and nine isomers or metabolites representing four insecticides were found in the urine. No endrin was found in any sample, even though the men had been exposed to it for an average of 2106 hr (Hayes and Curley, 1968).

Men engaged in the manufacture of endrin have significantly increased activity of hepatic microsomal enzymes. Hunter et al. (1972) assessed hepatic microsomal enzyme activity of workers engaged in the manufacture of pesticides by measuring changes in the urinary excretion of D-glucaric acid. Men employed in the manufacture of endrin alone had greater D-glucaric acid excretions than workers exposed to aldrin or dieldrin.

Low DDE [1,1-dichloro-2,2-bis(p-chlorophenyl)ethylene] levels found in endrin workers also suggest enzyme induction, since DDE is metabolized by microsomal enzymes (Hunter et al., 1972). A significant inverse relationship between the blood DDE level and urinary D-glucaric acid excretion is further evidence that this is the effect of enyzme induction.

Increased urinary concentrations of 6-beta-hydroxycortisol have also been reported in endrin workers (Hunter et al., 1972). This probably results from induction of the hepatic enzyme cortisol 6-beta-hydroxylase.

6.3.2 Acute Toxicity

Acute exposure can be defined as exposure to a test agent for 24 hr or less (National Academy of Sciences, 1975). Dosages in acute toxicity tests are usually selected to provide data for estimating the lethal dose for 50% of a group of animals and the slope of the dosage mortality curve. The LD_{50} is expressed as milligrams of endrin per kilogram of the weight of the test animal. Less toxic chemicals have larger LD_{50} values, since it takes a larger quantity to be fatal.

Endrin is classified as "very highly hazardous," meaning that any contact with very small amounts of the substance may result in severe injury or death (Thompson, 1971). Endrin is the most acutely toxic of the cyclodiene insecticides in use and yet, except for endosulfan, is least persistent in mammals (Brooks, 1974b). Endrin is toxic to all mammals regardless of route of exposure. When ingested in one dose by rats, endrin is about three times as toxic as aldrin and about 15 times as toxic as DDT (Treon et al., 1955). Upon intravenous administration in mice, endrin is five times as toxic as dieldrin (Walsh and Fink, 1972).

The onset of endrin toxicity symptoms is rapid. The return to normal among those who survive is also rapid. The recovery from endrin intoxication is quicker than from other cyclodiene insecticides (Weeks, 1967).

The symptoms of acute endrin poisoning in mammals clearly indicate that endrin is a neurotoxicant. The first indication of acute endrin poisoning is often central nervous system excitation evidenced by hypersensitivity to external stimuli associated with generalized tremors and followed by severe tonic-clonic convulsions (Brooks, 1974b). Convulsions may occur as soon as 30 min after acute endrin exposure (Weeks, 1967). Convulsions can culminate in death through respiratory failure (Brooks, 1974b). In the range of the acute oral LD_{50} (17 to 43 mg/kg), death of rats may result in about 48 hr (Boyd and Stefec, 1969).

Other symptoms of acute endrin poisoning include bradycardia (slowed heartbeat), increase in blood pressure, salivation, increased body temperature, leukocytosis (increased number of white blood cells), increased hemoconcentration, decreased blood pH, increased cerebrospinal fluid pressure and cerebral venous pressure, increased renal vascular resistance with decreased renal blood flow and glomerular filtration rate, decrease in catecholamine concentration of the adrenals, and increased levels of circulating epinephrine and norepinephrine (Emerson et al., 1964; Reins et al., 1966). Pulmonary edema was observed by Sen et al. (1969) in rats and mice. Pathological observations of rats at autopsy reveal signs of a stress reaction, degenerative changes in kidneys, liver and brain capillary and venous congestion, loss of weight, and dehydration of many organs (Boyd and Stefec, 1969).

The symptoms in man include headache, dizziness, abdominal disturbances, nausea, vomiting, mental confusion, muscle twitching, and epileptiform convulsions which may occur suddenly and without prior warning (Brooks, 1974b).

6.3.2.1 Oral toxicity. Mammalian susceptibility to endrin toxicity varies greatly with age, sex, and species, as shown in Table 6.2. The LD_{50} values range from 3 to 43 mg/kg.

Table 6.2. Acute oral toxity of endrin to mammals

Animal	LD_{50} (mg/kg)	References
Bats	5–8	Luckens and Davis, 1965
Rats (M)	17.8	Speck and Maaske, 1958
Rats (6 months, M)	43	Treon et al., 1955
Rats (6 months, F)	7	Treon et al., 1955
Rats (30 days, M)	30	Treon et al., 1955
Rats (30 days, F)	17	Treon et al., 1955
Rabbits (F)	7–10	Treon et al., 1955
Guinea pigs (F)	16	Treon et al., 1955
Guinea pigs (M)	36	Treon et al., 1955
Monkey	3	Treon et al., 1955

Sources: Luckens and Davis, 1965;
Speck and Maaske, 1958;
Treon et al., 1955.

It appears that monkeys are more susceptible than rats, and guinea pigs are more resistant. Rabbits appear to be somewhat more resistant than monkeys to a single dose of endrin. The acute toxicity of endrin is, however, high for all these species.

In rats and guinea pigs, females are more susceptible than males. The greater susceptibility of female rats six months of age than of younger female rats is the reverse of the more normal relationship between age and susceptibility in th case of males.

6.3.2.2 Dermal toxicity. When endrin, as a dry 100-mesh powder, was maintained in contact with either the intact or abraded skin of female rabbits for 24 hr, the minimum lethal dosage was found to be greater than 60 and less than 94 mg/kg. Poisoned animals had convulsions, but neither gross nor microscopic evidence of damage to the skin was found. Degeneration of the cells in the central zones of the lobules of the livers of rabbits was observed (Treon et al., 1955).

6.3.2.3 Other routes of administration. Graves and Bradley (1965) determined an LD_{50} of 5.6 mg/kg for endrin injected into the peritoneal cavity of Swiss albino mice. An intravenous LD_{50} value of 2.3 mg/kg was determined by Walsh and Fink (1972) for adult male mice. Endrin injected into dogs intravenously at a dosage of 3 mg/kg caused death in approximately 75% of the animals (Hinshaw et al., 1966).

6.3.3 Chronic and Subchronic Toxicity

Subchronic toxicity studies are designed to determine the adverse effects that may occur during repeated exposure over a period of a few days to about three months (National Academy of Sciences, 1975). Exposure levels in these studies are usually lower than in acute toxicity experiments. Studies involving repeated or continuous exposures to low dose levels for longer intervals could be considered chronic toxicity studies.

Target organs found in acute experiments are not always the same as those following repeated exposure over long periods of time. The central nervous system is the target of acute endrin poisoning. When an animal is exposed repeatedly to low doses (0.8 to 3.5 mg/kg/day) of endrin, it can often make compensatory adjustments to cope with the initial nervous system injury until damage to liver or other organ intervenes (Speck and Maaske, 1958).

After exposure of rats, rabbits, guinea pigs, cats, dogs, and monkeys to varying doses of endrin via several routes and frequencies of administration, diffuse degenerative changes were observed in the livers, kidneys, and brains of all fatally poisoned animals. Alternations were also observed in tissues of some of the surviving animals (Treon et al., 1955). Rabbits subjected to multiple dermal applications (20 to 44 mg/kg) exhibited severe fatty degeneration of the liver (Treon et al., 1955). A series of 50 oral doses of 1 to 5 mg/kg per day in a rabbit caused diffuse degeneration and fatty vacuolization of the hepatic and renal cells and degeneration of the heart. Rabbits that survived 118 periods of inhalation of 0.36 ppm endrin developed a granulomatous type of pneumonitis (Treon et al., 1955).

Chronic administration of endrin can lead to convulsions. Revzin (1970) administered endrin to squirrel monkeys at a minimum rate of 0.2 mg/kg per day, inducing characteristic change in the electroencephalogram (EEG) after relatively small total doses (0.5 to 1.0 mg/kg). At total doses of 5 to 10 mg/kg, electrographic seizures developed. Endrin administration was discontinued after seizures, but three weeks later, EEGs and behavior were still abnormal (Revzin, 1970). Recurrence of seizures, under stress conditions, months after termination of endrin administration demonstrates that even the small amounts of endrin stored in the body after exposure cannot be regarded as toxicologically inert (Revzin, 1970). Endrin mobilized by stress may cause toxic responses in the brain. If concentrations in fat in these monkeys (about 25 ppm after four months) were sufficient to cause seizures, the same stress to an animal storing 2.5 ppm may induce EEG and presumably behavioral changes (Revzin, 1970).

The chronic toxicity of endrin is greater than that of other organochlorine pesticides. In prolonged feeding experiments, rats can consume diets containing approximately three times as much aldrin and 12 times as much DDT as endrin without increase in the relative weight of specific organs. Dogs are at least ten times as susceptible to the toxic effects of endrin as to those of DDT (Treon et al., 1955). Species and sex differences exist in susceptibility to chronic endrin toxicity. Females are generally more susceptible than males. Rabbits and dogs are more susceptible than rats (Treon et al., 1955).

Exceptions to the usual higher susceptibility of females have been observed at low doses. Mortality rates observed by Nelson et al. (1956) indicate that male rats were significantly more susceptible to the toxic effects of endrin at the lower levels (1 and 5 ppm) than were females. Deichmann et al. (1970) found that mean survival rates for female rats fed 12 ppm endrin in laboratory chow was 18.2 months, while the mean survival of control rats were 19.5 months for females and 19.7 months for males.

Female rats appeared to be less susceptible than female rabbits but more susceptible than male rats to the cumulative action of endrin administered orally in multiple doses. Given an oral dosage of 1 mg endrin per kilogram of body weight on each of five days per weeks, four of five female rabbits died. This level of dosage (1 mg/kg) was tolerated by five female rats, but 2 mg/kg was fatal to two out of five female rats. Male rats survived when the level of dosage was 2 mg of endrin per kilogram, but died in the early period of the experiment when the level was 5 mg/kg (Treon et al., 1955).

6.3.3.1 Oral exposure. Mammalian species are sensitive to the toxic effects of endrin at low levels in the diet. Significant mortality over a seven-month period appeared in deer mice at 2 ppm in the diet (Morris, 1968). The deer mice showed symptoms of hypertension, incoordination, muscle tremors, and convulsions which increased in intensity until death occurred.

Endrin fed for a lifetime to Osborne-Mendel rats at 12 ppm in the diet decreased viability. Mean survival time fell from 19.7 months to 17.6 months for males and from 19.5 months to 18.2 months for females (Deichmann et al., 1970). Rats on the endrin diet experienced moderate increases in incidence of congestion and focal hemorrhages of the lung; slight enlargement, congestion, and mottling of the liver; and some enlargement, discoloration, or congestion of the kidneys (Deichmann et al., 1970).

Nelson et al. (1956) observed hypersensitivity to sound, touch, and pressure stimuli in all rats ingesting 1 to 100 ppm endrin for periods up to 16 weeks. In rats ingesting 25 ppm or more, the extreme sensitivity was followed by convulsions. Once convulsive patterns were established, rats rarely lived more than a week. Rats on a diet containing 25 ppm endrin also suffered from intermittent blindness, dysenteric symptoms, and slight bleeding through the nostrils. All rats receiving 100 ppm endrin died within two weeks. Of the ten rats that received 50 ppm, only two females survived the 16-week experiment (Nelson et al., 1956).

Groups of Carworth rats were given diets containing endrin in concentrations of 1, 5, 25, 50, and 100 ppm. The rats that died by the 80th and 106th weeks of feeding are shown in Table 6.3. Endrin in diets of female rats at levels of 25 ppm or higher caused significant mortality. Dietary levels of 50 ppm resulted in the early deaths of all but a few resistant rats. Male rats were less susceptible than female rats to the

Table 6.3. Mortality among groups of control rats and rats fed two years on diets containing endrin

Concentration (ppm)	No. that died/No. fed on diet			
	Males		Females	
	80 weeks	106 weeks	80 weeks	106 weeks
100	18/20	18/20	18/20	19/20
50	13/20	16/20	19/20	20/20
25	5/20	9/20	12/20	15/20
5	5/20	13/20	7/20	12/20
1	5/20	9/19	4/20	9/20
0	7/20	12/20	5/20	13/20

Source: Reprinted with permission from J. F. Treon, F. D. Cleveland, and J. Cappel, *J. Agric. Food Chem.* 3(10): 842-848 (1955). Copyright 1955 American Chemical Society.

Among rats fed on diets containing 50 to 100 ppm endrin, hypersensitivity to external stimuli and occasional convulsions were observed. These symptoms were not seen in those fed at 25 ppm (Treon et al., 1955). After rats had been fed diets containing endrin at levels of either 25 or 5 ppm for two years, the average ratios of the weights of their livers to their body weights were significantly greater than that of controls (Treon et al., 1955).

effects of endrin in the diet. A dietary level of 50 ppm caused a mortality increase over controls barely significant at the 5% level. At the dietary level of 100 ppm, however, only 5% of the males survived beyond two weeks. Rats that died exhibited diffuse degeneration of the brain, liver, kidneys, and adrenal glands. The incidence of neoplasia was no greater among the experimental animals than among the controls (Treon et al., 1955).

Dogs can consume safely about half the concentration of endrin in the diet that rats can tolerate. Dogs died when fed diets containing endrin in concentrations of 10 to 50 ppm (18 to 44 days). More than half of the dogs fed 5 to 8 ppm died. Dogs fed 10 ppm or more suffered extensive losses in weight. Dogs that died when fed on diets containing toxic concentrations (5 ppm or more) of endrin regurgitated their food, became lethargic, salivated, and later refused to eat. They became emaciated and developed respiratory distress and signs of irritation of the central nervous system (Treon et al., 1955).

Dogs fed 8 ppm endrin in the diet for almost six months exhibited enlargement of the liver, kidneys, and brain and reduction in the deposition of free peritoneal and omental fat. After dogs had been fed for almost 19 months on diets containing endrin at the level of 3 ppm, their kidneys and hearts were significantly enlarged (Treon et al., 1955).

Fatally poisoned dogs had diffuse degenerative lesions in the brain, heart, liver, and kidneys, together with pulmonary hyperemia and edema. Renal damage was severe and was characterized by the diffuse degeneration and neurosis of the convoluted tubules. The liver exhibited diffuse degeneration and fatty vacuolization and, in some instances, necrosis of the liver cells (Treon et al., 1955).

Speck and Maaske (1958) evaluated effects of chronic exposure by administering repeated oral doses (0.8, 1.7, and 3.5 mg/kg) of endrin daily, five days a week, to rats. Rats given these doses for one week had similar symptoms (red liver and contracted spleen) to rats receiving single high doses (20 to 80 mg/kg), but effects in animals receiving chronic doses were less severe. From three months up to the maximum tolerated exposure at seven months, there was a progressive decrease in color and an increase of spottiness of the liver, while spleens, blood, and lungs appeared normal. At the seventh month of repeated exposures to 3.5 mg/kg, there was a dramatic upsurge in the death rate (Speck and Maaske, 1958). Rats removed from a chronic exposure to endrin before irreversible liver damage occurred returned to normal. Rats removed from endrin exposure after three months showed no withdrawal symptoms (Speck and Maaske, 1958).

6.3.3.2 Inhalation. Following 7 hr of exposure on each of 130 days to air containing the sublimed vapor of endrin in the concentration of 0.36 ppm, two rabbits and a mouse died out of a group consisting of a cat, guinea pigs, hamsters, mice, rats, and rabbits. No convulsions were observed (Treon et al., 1955).

6.3.3.3 Dermal exposure. The intact skin of each of three female rabbits was maintained in contact with 150 mg dry endrin for 2 hr on each of five days per week. The rabbits died after 19, 19, and 25 such applications respectively (Treon et al., 1955).

6.3.4 Carcinogenesis and Mutagenesis

No malignancies attributed to endrin exposure have been found in the literature. Endrin fed to weanling Osborne-Mendel rats for a lifetime at levels of 2, 6, or 12 ppm in laboratory chow was neither tumorigenic nor carcinogenic (Deichmann et al., 1970; Deichmann and MacDonald, 1971; Deichmann, 1972a).

Endrin as well as aldrin and dieldrin can cause chromosome damage (Grant, 1973). Symptoms of cellular degeneration have been observed in germinal tissue of male albino rats treated with 0.25 mg of endrin administered intratesticularly (Dikshith and Datta, 1972). The most conspicuous effects were chromosomal aberrations, including stickiness, bizarre configurations, formation of chromosome fragments, and abnormal restitution of chromosomes. Formation of single and double bridges with acentric fragments was very common, disturbing the normal disjunction of chromosomes and eventually affecting the chromosome complements of the division products (Dikshith and Datta, 1973). Unequal distribution of chromosomes at anaphase was also observed. Severe cell damage resulted in liquefaction and transformation of the chromatin mass into an amorphous lump (Dikshith and Datta, 1972).

6.3.5 Reproduction and Teratogenesis

Endrin exposure appears to cause an increase in fetal and postnatal mortality, but fertility of the parents is generally not affected.

Endrin (5 ppm) in the diet of Sprague-Dawley rats for 60 days prior to conception and during pregnancy caused an increased number of resorptions and a decreased number of viable embryos (Green, 1967).

Good and Ware (1969) studied reproductive effects of endrin in Swiss mice. Virgin males and females were randomly paired. During four months of feeding 5 ppm endrin in the diet, adult mortality occurred in one-third of the pairs. Litters were significantly smaller than those produced by controls. The influence of endrin on reproduction is probably a direct effect on fetal mortality.

Postnatal mortality of young may be the main effect of endrin on reproductive performance in deer mice. Data of Morris (1968) suggest that average litter productivity of the deer mouse (*Perymys cus maniculatus*) is not adversely affected by endrin at doses up to 2 ppm in diet. Survival of parents was, however, significantly decreased at levels of 2 ppm or more.

Litter survival was adversely affected by feeding endrin to the parents. A severe postnatal effect on young was observed in deer mice whose parents ingested 4 or 7 ppm endrin in the diet.

If quantities of endrin sufficient to affect viability of young were to move across the placenta, the insecticide would first have to be moved from storage in the fat tissue into the blood stream. This movement might occur under conditions of parental stress or if stored levels reached a saturation point so that additional quantities would be circulated. Under these conditions, it is likely that parental death would precede parturition (Morris, 1968).

Endrin exerted embryocidal and teratogenic effects on pregnant hamsters (Ottolenghi et al., 1974). Both soft and skeletal tissue malformations were produced. Single oral doses of endrin (5 mg/kg) administered to pregnant Syrian golden hamsters on day 7, 8, or 9 of gestation caused a high incidence of fetal death, congenital abnormalities, and growth retardation. Thirty-two percent of the implantations resulted in fetal mortalities. Teratogenic effects were observed in 28% of the fetuses from hamsters treated on day 8. Open eye occurred in 22%, webbed foot in 16%, cleft palate in 5%, cleft lip in 1%, and fushed ribs in 8%.

Endrin is teratogenic in mice, but frequency and gravity of the defects produced were less pronounced than in the hamsters when a single oral dose (2.5 mg/kg in mice and 5 mg/kg in hamsters) of half the LD_{50} was administered. Abnormalities in mice included open eye and cleft palate (Ottolenghi et al., 1974).

The only reported effect on fertility was caused by a single administration of endrin into the rat testes, which produced hypertrophic changes in the testis and induced total arrest of spermatogenesis. Endrin action was not specific to any group of spermatogenic cells; rather, the entire tubular and intertubular areas suffered total and irreversible damage. All the spermatogenic cells of the tubules revealed massive necrosis. Seminiferous epithelium and cellular architecture of the tubules were totally altered, and the necrotic cells coalesced to form a mass (Dikshith and Datta, 1972).

6.3.6 Physiological Effects

6.3.6.1 Nervous system. The toxicity of endrin to humans appears to result primarily from the effect of endrin on the central nervous system. The major clinical manifestation of endrin intoxication in humans is convulsive seizures of several minutes duration followed by semiconsciousness (Weeks, 1967; Cobel et al., 1967; Jacobziner and Raybin, 1959). Convulsions and hypersensitivity are probably caused by endrin acting directing on the motor cortex and/or spinal cord (Emerson et al., 1964).

Convulsions usually occur in humans within 2 to 4 hr following ingestion of endrin-contaminated food. Children are less likely to have convulsions from acute poisoning than are adults, but there is a wide range of sensitivity in both adults and children.

In rats, a latent period of 45 to 60 min elapsed before the appearance of convulsions regardless of size of an effective single dose (20 to 80 mg/kg) (Speck and Maaske, 1958). The data suggest that the concentration of endrin had to reach a critical level in the brain and/or other tissues for convulsions to occur. Severe clonic convulsions were apt to be set off by touch or auditory stimuli. Some rats survived repeated severe attacks and showed catatonic behavior for 30 min afterward (Speck and Maaske, 1958). Severe tonic-clonic convulsions in dogs commenced within five to ten min after beginning intravenous infusion with 10 mg/kg endrin (Emerson et al., 1964). Convulsions, as well as hyperexcitability to stimulation, and copious, mucoid salivation lasted until death. Symptoms manifested were similar to those observed in humans. Death occurred from respiratory failure.

Electroencephalogram changes frequently precede convulsions, and withdrawal from exposure usually results in a normal EEG within one to six months (Cobel et al., 1967). During endrin exposure, the EEG may show bilateral synchronous spikes, spike-and-wave complexes, and slow theta waves.

Seven daily injections (0.2 mg/kg) of endrin in the squirrel monkey caused increases in EEG amplitude and a tendency toward increased spiking. These changes became more marked as a function of total dosage until seizure activity developed. The EEG remained abnormal for more than one month after administrations were terminated (Revzin, 1968).

Joy (1976) injected cats with 1 to 2 mg/kg endrin and found that spontaneous seizures occurred within 5 to 15 min. Higher doses produced seizures in 0.5 to 2 min. Some test animals developed transient hypotension followed by hypertension and arrythmias. EEG changes were clearly defined and consistently present. Following the administration of endrin, hypersynchrony, rhythmic bursts of high-voltage spikes, and isolate spikes invariably developed immediately prior to the seizure. Seizures were bilateral and symmetrical and of a tonic-clonic type. Evolution of the entire EEG sequence leading to a seizure was found to occur within 2 min after injection of endrin.

EEG recordings made on rats after an 80 mg/kg dose of endrin showed irregular slowing, some irregular spikes, and frequent convulsive discharges (Speck and Maaske, 1958). EEG changes were minimal, however, in rats receiving chronic doses of 3.5 mg/kg or less. After one week of exposure to endrin, some rats had bursts of multiple spikes accompanied by clonic convulsions, and additional runs of spikes without full-fledged convulsions. Convulsions were usually preceded by a period of hyperventilation. After two weeks of exposure, the rats had normal EEGs until liver damage began to appear after three months of repeated doses. At three months the animals again became susceptible to audiogenic convulsions. At seven months, convulsions were easily started (Speck and Maaske, 1958). Rats withdrawn from chronic exposure were no longer susceptible to convulsion after seven weeks of withdrawal (Speck and Maaske, 1958).

Endrin can affect central nervous system processing at doses substantially below lethal or convulsive levels. Consequently, behavior can be affected in the absence of the tremors and seizures generally considered signs of endrin poisoning. Revzin (1970) observed EEG changes in a squirrel monkey receiving a total endrin dose of 1 mg/kg. Seizures were not observed until the total dose reached 10 mg/kg. Endrin levels in the general human population, however, are probably not sufficient to induce changes in brain functions (Revzin, 1970).

Other symptoms of endrin toxicity may result indirectly from the effect of endrin on the central nervous system. Convulsions may interfere with normal respiratory exchanges of blood gases, resulting in the acidosis and hyposia found consistently in endrin poisoning (Hinshaw et al., 1966). Action of endrin on the central nervous system seems to indirectly affect both sympathetic and parasympathetic nervous systems and the adrenals (Hinshaw et al., 1966). Stimulation of parasympathetic nerves may cause bradycardia and excessive salivation, while the sympathetic nerves may be responsible for other cardiovascular symptoms in endrin intoxication (Fig. 6.2).

6.3.6.2 Cardiovascular effects. Endrin poisoning in mammals is accompanied by marked cardiovascular effects, including bradycardia, hypertension, increased venous return and corresponding elevation in cardiac output, and decreased renal blood flow. Most of these cardiovascular effects appear to be caused indirectly by endrin acting on the central nervous system (Fig. 6.2). The symptoms suggest that both sympathetic and parasympathetic nervous systems are hyperactive after endrin

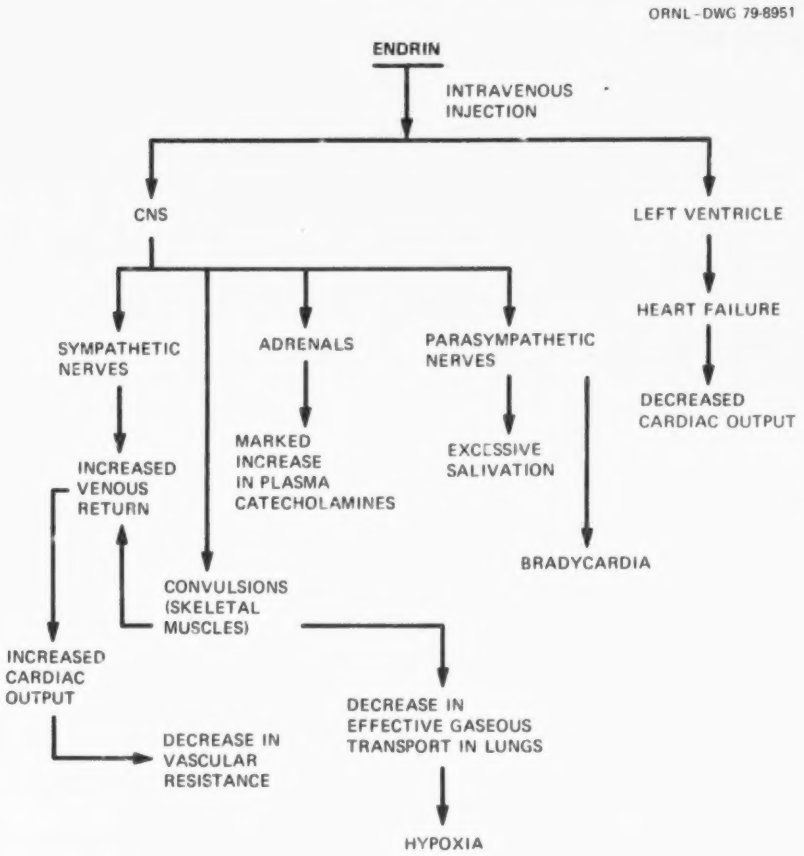

ORNL-DWG 79-8951
ENDRIN
INTRAVENOUS INJECTION
CNS
LEFT VENTRICLE
HEART FAILURE
DECREASED CARDIAC OUTPUT
SYMPATHETIC NERVES
ADRENALS
PARASYMPATHETIC NERVES
INCREASED VENOUS RETURN
MARKED INCREASE IN PLASMA CATECHOLAMINES
EXCESSIVE SALIVATION
BRADYCARDIA
CONVULSIONS (SKELETAL MUSCLES)
INCREASED CARDIAC OUTPUT
DECREASE IN EFFECTIVE GASEOUS TRANSPORT IN LUNGS
DECREASE IN VASCULAR RESISTANCE
HYPOXIA

Fig. 6.2. Suggested schema for physiological changes produced by endrin. Source: Hinshaw et al., 1966.

administration. Stimulation of the parasympathetic nerves would cause bradycardia. When the sympathetic nervous system throughout the body is stimulated to cause direct effects on the blood vessels, it also causes the adrenal medullae to secrete catecholamines (epinephrine and norepinephrine). These hormones then circulate everywhere in the body fluids and act on all vasculature (Guyton, 1971). Dogs given endrin exhibited large mean increases in catecholamine concentration from less than 4 μg per liter of plasma to over 55 mg/liter (Hinshaw et al., 1966). Adrenalectomy significantly decreased catecholamine concentrations, which, however, were significantly elevated above pre-endrin values (Hinshaw et al., 1966). The slight, though significant, elevation in total plasma catecholamines after endrin exposure in adrenalectomized dogs suggests the active participation of the sympathetic nervous system. Many sympathetic postganglionic axons are adrenergic fibers, capable of releasing norepinephrine and smaller amounts of epinephrine (Hinshaw et al., 1966). Increases in levels of epinephrine and norepinephrine in the blood with probable parasympathetic activation make it difficult to evaluate the exact role of catecholamines in the cardiovascular effects of these compounds

(Reins et al., 1966). These high levels of epinephrine and norepinephrine are probably enough to increase systemic arterial pressure through increased cardiac output following elevation of venous return. High levels of epinephrine apparently do not increase heart rate because of an overriding reflex vagal effect (Reins et al., 1966).

Increased systemic arterial pressure has been observed as an early effect of a lethal dose of endrin administered to dogs immobilized with succinylcholine (Reins et al., 1964). This rise in systemic arterial pressure depends primarily on increased cardiac output. Endrin does not have a direct excitant action on the heart to cause an increase in cardiac output, thus elevating venous return. The increased volume of blood returning to the right atrium of the heart after endrin exposure appears to originate primarily in the abdominal viscera (Reins et al., 1966). Release of stored blood results in a continued maintenance of a high cardiac output. Since the released blood remains in the active circulation, venous return is continually maintained at a higher level. The increase in venous return is not induced by circulating catecholamines, since adrenalectomy does not modify the response (Hinshaw et al., 1966). Systemic peripheral resistance markedly falls with endrin administration, which could be due to the marked rise in blood flow. Total peripheral resistance is ordinarily related inversely to blood flow (Hinshaw et al., 1966). Total vascular resistance represents net changes in resistance. Some vascular beds may be dilating and some constricting. Dogs administered 10 mg/kg endrin showed marked renal vasoconstriction and, therefore, elevated resistance in the renal bed (Reins et al., 1964).

Emerson and Hinshaw (1965) investigated the effects of endrin on vascular resistances in forelimbs of anesthetized dogs. Endrin administration resulted in large increases in total limb vascular resistance, most of which were due to small-vessel (small artery to small vein) resistance. The arterioles constitute a major resistance to blood flow. Arterial and venous segmental resistances also increased. Sympathetic innervation of limb vessels is not necessary for the large resistance increases which follow endrin administration. The resistance patterns of the innervated and denervated groups are similar for the first 10 to 15 min after endrin exposure (Emerson and Hinshaw, 1965). Resistance then increases considerably more in denervated limbs than in innervated.

Increased vascular resistance in the kidney following endrin infusion (10 mg/kg body weight) has been shown to be due primarily to circulating catecholamine-like agents, but renal nerves also play a role (Reins et al., 1964).

In addition to cardiovascular effects exerted through the central nervous system, endrin appears to exert a toxic action directly on the left ventricle (Fig. 6.2). Left ventricle failure demonstrated by elevated left atrial pressure occurred regularly in endrin-treated dogs (Hinshaw et al., 1966). Cardiac standstill occurred in more than 20% of the dogs. It is not understood why endrin has a damaging action on the left ventricle, while the right ventricle appears to be unaffected.

6.3.6.3 Enzyme systems. Some symptoms of endrin intoxication may arise through effects of endrin on mammalian enzyme systems.

Metabolic effects of endrin are particularly likely to be influenced by change in enzyme levels. Metabolic changes after endrin exposure have been indicated by weight loss and by changes in both oxygen uptake and carbon dioxide production.

Nelson et al. (1956) observed a significant relation between endrin consumed and weight change at various time intervals. Rats on 100 ppm lost the greatest weight followed by those on 50 and 25 ppm. A greater weight loss was found in male rats than in females. Although the total average feed consumption of rats receiving endrin was less than controls, a reduced food intake was not entirely responsible for the weight loss. Even where control and experimental animals consumed the same amount of food, the endrin-fed animal lost considerably more weight than its control.

Marked increases in both oxygen uptake and carbon dioxide production resulted from intravenous injection of 3 mg/kg endrin into dogs. Mean respiratory quotient values, however, remain relatively unchanged. Arterial and venous concentrations of oxygen are very low within 30 min after administration of endrin (Hinshaw et al., 1966).

The ability of a single dose of endrin to significantly enhance the potential of kidney cortex and liver to synthesize glucose from non-carbohydrate sources in animals has been demonstrated (Singhal and Kacew, 1973). Alterations in carbohydrate metabolism produced by endrin may be related to an enhanced capacity of liver and kidney cortex to synthesize cyclic AMP (adenosine monophosphate). The cyclic AMP-adenyl cyclase system is believed to play an important role in the action of several chemicals on carbohydrate metabolism. A single oral dose of endrin (50 mg/kg body weight) to rats stimulated the activities of the following key rate-limiting enzymes involved in the pathway of gluconeogenesis: renal and hepatic pyruvate carboxylase, phosphoenolpyruvate carboxykinase, fructose,1,6-diphosphatase, and glucose 6-phosphatase. In addtion, endrin depressed liver glycogen and elevated the concentration of serum glucose and urea (Kacew and Singhal, 1973a; Kacew et al., 1973; Singhal and Kacew, 1976).

When kidney cortex homogenates, kidney slices, or liver slices were incubated with endrin in vitro, a significant stimulation of cyclic AMP formation from radioactive adenosine was noted (Kacew and Singhal, 1973b; Singhal and Kacew, 1973). Administration of exogenous cyclic AMP was found to mimic the action of endrin on gluconeogenous and glycogenolysis as well as the hyperglycemic and hyperuremic responses (Singhal and Kacew, 1973). Since endrin-induced changes in carbohydrate metabolism are similar to those produced by exogenous cyclic AMP, endrin-induced changes in carbohydrate metabolism of kidney and liver might involve alterations on the cyclic AMP system.

Endrin may affect major components of the terminal oxidation system. The possibility that endrin exerts an effect on electron transport enzymes by its ability to associate with lipoprotein components of the mitochondrion raises the question of whether the phosphorylative capacity of endrin-treated mitochondria is altered. Preliminary experiments on phosphorylation in rat liver mitochondria treated with endrin revealed that ATP formation, measured as radioactive-labeled PO_4 incorporation into glucose-6-phosphate in the presence of excess hexokinase, was inhibited up to 10% (Colvin and Phillips, 1968).

Colvin and Phillips (1968) suggest that any inhibition by endrin of the transmission of nerve impulses would be due to binding of endrin to lipoprotein components essential for mitochondrial oxidation and not directly due either to inhibition or stimulation of acetylcholinesterase. They propose that endrin and its analogues bind to lipid-rich structural components of mitochondrial and certain other organelles. This binding may subsequently affect the activity of enzymes associated directly with the lipid-rich fractions.

A single intraperitoneal injection of endrin (10 mg/kg) significantly inhibited the total lipid content of the liver form 12 to 48 hr after treatment. An increase in total liver weight, however, indicated increased protein synthesis. Kidney protein levels were not affected, but glycogen levels were increased. Therefore, when rats are exposed to single doses of endrin, the total lipid and phospholipid levels are decreased, but there is no significant change in cholesterol levels (Gupta and Kaushal, 1977; Kaushal and Gupta, 1977).

Pardino et al. (1971) investigated the effect of endrin on in vitro, mitochondrial electron transport. Endrin was found to be a marginal inhibitor of the beef heart mitochondrial succinoxidase system and the beef heart mitochondrial-reduced nicotinamide adenine dinucleotide-oxidase system. Endrin depressed the activity of both enzyme systems to approximately 80% of controls. Other evidence indicates that endrin induces hepatic microsomal enzymes. Measurements of urinary 6-beta-hydroxycortisol in workers exposed to endrin indicate that endrin can induce hydroxylating enzymes of hepatic microsomes (Chamberlain, 1971). Levels of 6-hydroxylating enzyme activity in the group exposed to endrin were significantly higher than in control groups or a group exposed to aldrin and dieldrin.

Endrin causes an increased serum alkaline phosphatase level. When various levels of endrin (0, 1, 5, 25, 50, and 100 ppm) were added to a basal diet, the serum alkaline phosphatase levels were higher among rats consuming endrin than in the control group, and this increase was related to the quantity of endrin consumed during a period of time (Nelson et al., 1956). The liver plays an important role as a source of alkaline phosphatase. Elevated serum alkaline phosphatase levels have been associated with functional impairment of the liver and also with abnormalities in dephosphorylation mechanisms (Nelson et al., 1956).

The administration of a single oral convulsive dose (20 mg/kg) of endrin to albino mice significantly increased serum enzyme levels of glutamic oxaloacetic transaminase, glutamic pyruvic transaminase, and lactic dehydrogenase (Luckens and Phelps, 1969). Serum enzyme activity levels appear to be directly related to cellular damage. Damaged organs and tissues have been found to show a decrease in enzyme activity. The proportions of enzymes from such organs agree with those concomitantly found in the serum (Luckens and Phelps, 1969). This may account for the fact that although Luckens and Phelps (1969) observed an increase in serum lactic dehydrogenase upon endrin exposure, Hendrickson and Bowden (1973, 1976) found endrin inhibiting to rabbit muscle lactic dehydrogenase.

6.3.6.4 Hematology. Various changes in blood composition have been observed following endrin exposure. Endrin appears to cause hemocentration as evidenced by increases in hematocrit (the percentage of total blood volume consisting of erythrocytes) and in leukocyte concentration.

Dogs given lethal doses of endrin (30 mg/kg) exhibited large increases in the concentration of leukocytes (Emerson et al., 1964). Both mobilization of stored leukocytes and increased bone marrow production appear to have contributed to the increased plasma leukocyte concentration. Hemoconcentration was observed in most animals. The trend toward acidosis during endrin intoxication did not appear to be of respiratory origin, since increasing ventilatory rate did not prevent the decreased pH (Emerson et al., 1964).

Hematocrit increases in dogs were observed during acute endrin poisoning (Emerson, 1965). A hematocrit increase can be due to increase in size or number of erythrocytes. Emerson (1965) showed that approximately half the postendrin rise in hematocrit is prevented by removal of the spleen. This indicates the expulsion of cell-rich blood from spleen storage is responsible for an appreciable portion of hematocrit elevation. Other mechanisms must also be involved, however, to account for the hematocrit elevation. Other mechanisms must also be involved, however, to account for the hematocrit increase in splenectomized dogs. Loss of filtration is possible. Severe acidemia could lead to an increased hematocrit subsequent to an increased erythrocyte size (Emerson, 1965).

Coleman (1968) analyzed blood serums of rats given daily intraperitoneal endrin doses (1 mg/kg). No changes were found in general serum protein, serum lipoproteins, albumin, and in alpha, beta, or gamma globulins. Protein-bound sialic acid, methyl pentose, and bound hexose rose to above normal levels. Bound hexosamine was decreased. Alterations in bound carbohydrates would indicate metabolic alterations in serum glycoproteins.

6.3.6.5 Trace-metal mobilization. Endrin intoxication produces, in the initial stages, significant changes in concentration of metals in various organs (Coleman et al., 1968). Shifts in certain trace metals at the molecular level may be related to observed symptoms of endrin intoxication (Lawrence et al., 1968). Such trace-metal shifts resulting from endrin exposure might precede and contribute to gross physiological manifestations of endrin intoxication. Metal concentrations in organs, tissues, and body fluids of rats were studied under conditions of actue endrin intoxication by Lawrence et al. (1968) and under conditions of subacute endrin intoxication by Coleman et al. (1968).

Lawrence et al. (1968) exposed rats to a single oral dose of 25 mg of endrin per kilogram body weight. The rats experienced increases in size of all organs except the spleen within 2 hr of administration. Increases were greatest for the liver and heart. The heart and spleen demonstrated the greatest mobilization of metals. In both organs the total amounts of iron, magnesium, and zinc increased, while the total amount of copper decreased. Changes in unit concentrations paralleled those of the total organ contents, indicating trace-metal shifts over and above that attributable to change in the organ size parameters.

The kidney was intermediate in metal translocation, demonstrating significant increases in concentration of iron, magnesium, and zinc.

The principal trace-metal shifts experienced by the brain and liver included decreases in both total and unit concentrations of magnesium and copper in the brain, and of copper in the liver. These decreases occurred in opposition to increases in sizes of both of these organs. Total amounts of zinc in brain and liver and iron in liver increased, while unit concentrations did not change significantly.

Zinc exhibited increases in the total organ content of heart, liver, brain, spleen, and kidney. An observed decrease in zinc in red blood cells provides one possible source of the mobilized zinc.

Copper concentrations and total amount of copper present decreased in all organs studied except the kidney. The appearance of increased plasma levels of this metal indicates that copper was mobilized toward the circulatory system.

Magnesium increased in kidney, spleen, and heart. The increases in unit concentrations, accompanied by increases in size of kidney and heart, indicate extreme migration of magnesium into these organs. Decreased magnesium levels observed in the brain, red blood cells, and plasma suggest that magnesium was mobilized at the expense of the circulatory and central nervous systems.

Total iron content, as well as unit concentration, increased in kidney, spleen, and heart. Total iron content of liver increased without significant change in unit concentrations.

Coleman et al. (1968) investigated the possibility that mobilization of some of the most biologically significant metals (iron, magnesium, zinc, and copper) occurs in those organs, tissues, and body fluids most responsive to endrin exposure.

After eight daily sublethal exposures (1 mg/kg body weight per day), the number of trace-metal translocations was small compared with those occurring during the initial intoxication, indicating a tendency of most organs to recover. Two exceptions to this trend were concentrations of magnesium in the liver and copper in the kidney, both of which remained essentially unaltered during the initial stages of intoxication but later increased.

Under acute exposure (single oral dose of 25 mg/kg body weight) the spleen demonstrated significant changes in all metals studied, but no changes in organ size (Lawrence et al., 1968). After eight daily exposures, the spleen decreased drastically in size, but the number of metal shifts was comparatively small. Subacute oral exposure of rats to multiple doses of endrin (1 mg/kg body weight per day) produced, over an eight-day period, effects on organs differing from effects observed in an earlier acute study (Lawrence et al., 1968) in the same laboratory. In the subacute, a decrease in size of spleen was the only significant change (Coleman et al., 1968). In the acute study the spleen was the only organ that did not experience a significant change in size.

Significant increases in excretion of magnesium and decreases in excretion of zinc in the urine and feces accompanied endrin intoxication (Coleman et al., 1968).

6.3.6.6 Liver. Endrin produces liver damage similar to that seen after poisoning by other chlorinated hydrocarbons.

After an acute lethal dose of endrin (10 mg/kg body weight), dogs exhibited mild to moderate congestion and irregularly distributed cloudy swellings in the liver (Reins et al., 1964). Elevated serum alkaline phosphatase levels associated with addition of various levels of endrin to the diet of rats may indicate functional impairment of the liver (Nelson et al., 1956).

A major difference between acute and chronic endrin exposure manifests itself in effects on the liver. Rats receiving chronic low doses developed zones of basophilic cells around the central and portal veins. These abnormal cells were seen as early as seven days and increased in severity and incidence as exposure was increased. The livers of acute animals showed no discernible microscopic changes (Speck and Maaske, 1958).

6.3.6.7 Kidney. Studies by Reins et al. (1964) offer no evidence that endrin has a direct toxic effect on the kidney. Renal alterations appear to result secondarily from hemodynamic effects of circulating humoral agents, systemic hypertension in acute poisoning, and terminal systemic hypotension in animals with chronic exposure.

Dogs given acute doses of endrin (10 mg/kg) by intravenous infusion developed increased renal vascular resistance largely attributable to sympathoadrenal action. Only a small increase in renal resistance was seen in adrenalectomized animals. Acute effects of endrin were predominantly afferent arteriolar vasoconstriction as evidenced by decreases in renal blood flow, glomerular filtration rate, and urine flow. Although renal vascular resistance appears to be due primarily to circulating catecholamines, renal sympathetic nerves have a minor but variable role in control of resistance of the renal vasculature (Reins et al., 1964).

Changes in renal function of dogs in chronic studies were minimal and appeared to be due to secondary alterations in systematic hemodynamics. Dogs received 1-mg/kg doses of endrin five days a week until death (Reins et al., 1964). No definite tubular changes were detected in the kidneys.

6.3.6.8 Spleen. After an acute lethal dose of endrin (10 mg/kg body weight), dogs exhibited moderate to severe congestion of the spleen, with early degenerative changes and a mild degree of necrosis in the reticular cells associated with the germinal centers. Degenerative changes were more prominent in the large active germinal centers (Reins et al., 1964).

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7. MEDIA DISTRIBUTION, TRANSFORMATION, AND TRANSPORT

7.1 SUMMARY

Endrin was introduced in the United States in 1951 and is manufactured both domestically and abroad. Its primary use is as an insecticide, and its major application is to cotton. A wide variety of other crops may also be protected from insect pests by endrin. The most recent year for which consumption data are available is 1971. In that year a total of 643,772 kg (1,418,000 lb) of endrin was used. This value represents a significant decrease from the 1964 figure of 984,726 kg (2,169,000 lb).

Endrin enters the environment as a result of direct application to soil and crops. The persistence of endrin in the soil is, however, dependent upon so wide a variety of factors that half-lives ranging from 24 hr to 11.8 years have been reported. Included among these factors are properties of the soil, agricultural processes, topography, and numerous meteorological determinants. Soils contaminated by endrin are located predominantly in the cotton belt or delta states (such as Arkansas, Louisiana, and Mississippi), where endrin is used extensively. Concentrations as high as 0.64 ppm were observed in Mississippi soils during 1972, while maximum concentrations in Arkansas and Louisiana soils during 1973 where 0.24 and 0.48 ppm respectively.

Many pathways have been established for the removal of endrin from the soil. Mechanisms such as volatilization, leaching into groundwater, wind erosion, and surface runoff result in the translocation of endrin to other compartments of the environment, namely, the atmosphere and hydrosphere. Other pathways, including photodecomposition, thermal decomposition, and microbial degradation actually result in the dissipation of endrin from the environment as well as from the soil.

The major source of endrin in rivers and other freshwater bodies is surface runoff from fields and crops following application. Factors similar to those affecting the persistence of endrin in the lithosphere also influence the extent to which surface runoff will occur. Where intense precipitation falls on sites of low permeability and low organic content, runoff usually occurs. Steep topography accentuates runoff losses.

Additional sources of endrin in the hydrosphere are runoff from endrin-coated seeds, contaminated industrial effluents, contaminated fallout and precipitation, and carelessness in application and handling. Pesticide surveillance of the surface waters of United States river basins has been in effect since 1957. From 1961 through 1964 the number of samples containing endrin progressively increased until, in 1964, 50% of all samples tested were contaminated. Consistent contamination over the years of a large percent of the samples occurred only in the lower Mississippi basin. In addition, the highest concentrations recorded during the six consecutive years from 1960 to 1965 were all from this area. Endrin contamination of the lower Mississippi is apparently declining. While the residue levels reported between 1970 and 1975 were significant (20 ppt), they did not approach the maximum of 214 ppt present in 1963 when numerous endrin-related fish kills occurred.

Removal of endrin from the hydrosphere occurs by several routes. Volatilization and codistillation, although extremely significant, merely involve a reallocation of the endrin to the atmosphere. True dissipation occurs as a result of biochemical and photochemical degradation. While degradation of endrin by aquatic microorganisms has been reported, no studies in support of photochemical decomposition in the hydrosphere are available. It is believed, however, that such decomposition does take place to some extent.

Endrin adsorbs to suspended particulates in the hydrosphere and is transported to the sediment. The large-scale use of endrin in the agricultural lands surrounding the Mississippi River basin prior to 1964 was reflected in the levels of the pesticide detected in bottom deposits (10,000 ppt) in that year. The highest concentration of endrin in the sediment of any river basin between 1970 and 1975 was also found in the lower Mississippi. This value, 6700 ppt, was somewhat less than that reported in 1964. Decreased input, resuspension of sediment following turbulent periods (such as storms), and some transport of contaminated sediment to the sea are probably all responsible.

Vaporization of endrin from treated soils and crops is believed to represent the major source of atmospheric contamination. In one experiment, only 6% of the endrin sprayed on cabbage plants was detected in soil or leaves four weeks following application. Codistillation from water, wind transport of pesticide-laden dust, aerial drift from spraying operations, and vapor emissions from industrial sources also contribute to the presence of endrin in the atmosphere. Endrin is present in the atmosphere both in the vapor phase and adsorbed to wind-borne particulates.

Extensive monitoring for organochlorine pesticides in the atmosphere has not been conducted, and data for endrin are limited. From what little data are available, it is apparent that concentrations are highest in the agricultural centers of the delta states, where use of endrin predominates. The highest concentration reported to date was 58.5 ng/m^3 in Stoneville, Mississippi, in 1971. Urban communities far removed from agricultural areas are unlikely to experience significant contamination. The majority of organochlorine insecticides released to the atmosphere are eventually returned to the earth's surface. While some dissipation in the atmosphere via photodecomposition is possible, no real evidence that such transformation actually takes place is available. Therefore the major hazard involved in release of endrin to the atmosphere is the subsequent contamination of the hydrosphere and lithosphere. The atmosphere transports far larger quantities of organochlorine insecticides than does any other mechanism, and atmospheric transport is considered to be primarily responsible for the worldwide dissemination of these poisons. Although endrin per se has not been detected in remote areas, the structural, chemical, and toxicological similarities between endrin and related compounds detected at great distances from their point of application suggest that similar patterns of transport may be operative.

Transport of endrin to the open ocean is expected to occur primarily via the atmosphere. While river transport of contaminated, suspended particulate matter may also contribute, codistillation and sedimentation would probably remove most of the endrin from the flowing water long before it reached the sea.

Atmospheric endrin is conveyed to the ocean by particle fallout or precipitation washout. Upon arriving at the surface of the sea, the endrin may adsorb to the organic materials present in the surface microlayer and be subject to revolatilization, or it may adhere to large suspended particulates and be carried to the sediment. Due to the depth of the ocean, the chances for resuspension of contaminated sediment are, at best, limited. The sediment of the oceanic abyss therefore represents the ultimate sink for endrin and related compounds. The fate of endrin in the ocean sediment remains unknown.

7.2 PRODUCTION AND USE

Endrin was introduced in the United States in 1951 by the Hyman Company and is manufactured domestically by the Velsicol Chemical Corporation and in the Netherlands by Shell Nederland Chemie. The only known uses of endrin are as an avicide, a rodenticide, and an insecticide, the last of these being the most important. The endrin sold in the United States is a technical-grade product containing not less than 95% active ingredient. It is available in a variety of diluted formulations including wettable powders, dusts, emulsifiable concentrates, granules, oil solutions, and baits (Shell Chemical Corporation, 1959; International Agency for Research on Cancer, 1974).

Endrin is approved for use in the protection of forest seeds against birds, mice, and chipmunks, and for the control of birds on buildings and mice in orchards (International Agency for Research on Cancer, 1974). As an insecticide, its major application is to cotton crops, which it protects from sucking, leaf-eating, and boll-damaging varieties of insects throughout the growing season (Brooks, 1974a). Insects that attack a wide variety of vegetables, field crops, fruits, nuts, and pasture are also controlled by endrin (Eichers et al., 1968; Eichers et al., 1970; Adrilenas, 1974).

The use of endrin is apparently declining. In 1964, endrin was included among the 12 leading insecticides with a total consumption of 2,169,000 lb. By 1971, consumption had decreased to approximately 65% of the 1964 value, or to 1,427,000 lb (Eichers et al., 1968; U.S. Department of Agriculture, 1968; Andrilenas, 1974). The current consumption figures are not available. Endrin usage has been banned in Switzerland, Italy, and Germany (International Agency for Research on Cancer, 1974).

7.3 ENDRIN IN THE ATMOSPHERE

7.3.1 Introduction

Evidence suggests that more than half of the organochlorine insecticides sold annually eventually enter the atmosphere (Frost, 1969). Although photochemical decomposition of many of these insecticides does take place, even generous allowances for this factor result in the conservative estimate that thousands of tons are transported to the oceans via the atmosphere each year (Frost, 1969). The atmosphere transports far larger quantities than any other mechanism, and atmospheric transport is considered to be primarily responsible for the worldwide dissemination of these poisons. In order to control the escape of organochlorine insecticides into the atmosphere, it is first necessary to understand the mechanisms by which such escape takes place. Losses are known to occur by several routes, including: volatilization from soil and crops, evaporation and codistillation from contaminated bodies of water, wind transport of pesticide-laden soil and dust, aerial drift from spraying and dusting applications, venting of vapors from pesticide manufacturing and formulating plants, and open burning of discarded pesticide containers at inadequate temperatures.

All of the above routes have not been investigated for every organochlorine insecticide. However, the structural, chemical, and toxicological similarities among these compounds (especially the chlorinated cyclodienes) suggest that similar patterns of loss may be expected. The number of experimental studies dealing specifically with loss of endrin to the atmosphere are adequate in some areas but nonexistent in others. Where experimental data are lacking for endrin, but are sufficient to implicate a given route to the atmosphere as significant for a related compound, this route must be considered as a potential source of endrin as well. Only after studies have definitely ruled out a mechanism is it safe to assume that the mechanism does not apply.

In interpreting what little experimental evidence is available, care must be taken to assure that release to the atmosphere has actually been observed. Studies dealing with "disappearance" of endrin from soil and crops are particularly suspect. Endrin may "disappear" for other reasons as well. Photodecomposition, bacterial degradation, absorption by plants, etc., must also be taken into account. If an analytical procedure does not extend to the decomposition products as well as to endrin itself, then one cannot be certain that vaporization is wholly responsible for loss.

Monitoring data for ambient atmospheric endrin concentrations have also been reported. Data, however, are scarce and generally not totally reliable. Analytical difficulties, problems associated with monitoring sufficiently large volumes of air, failure to trap vapors as well as particulate matter, interfering compounds, and equipment failure are all responsible. However, some feeling for the extent and degree of atmospheric contamination by endrin is provided by these data, and despite their inaccuracies they seem worthy of presentation.

7.3.2 Sources of Endrin in the Atmosphere

7.3.2.1 Vaporization from soil and crops. Vaporization of endrin from treated soils and crops is believed to represent the major source of atmospheric contamination (Abbott et al., 1966; Frost, 1969; Chesters and Konrad, 1971; U.S. EPA, 1972). Some idea of the evaporative potential of endrin under controlled laboratory conditions is provided by the work of Bowman et al. (1965). Samples of endrin (375 mg in 4 ml of hexane) were placed in glass beakers with a bottom surface area of 8 cm^2 and incubated in a gravity convection-type oven at 45°C. This temperature was arbitrarily chosen as the highest temperature to which soils in a southern state (Georgia) are likely to be exposed under field conditions. Samples were analyzed for endrin and possible degradation products by electron-capture gas chromatography following four and eight days of incubation. After four days, 38% of the endrin had volatilized; eight days of incubation resulted in the loss of 82% of the initial quantity. No degradation products were observed under the conditions of this experiment.

Studies conducted in the field appear to confirm the expected rapid loss of endrin to the atmosphere following application to soil and/or crops. Volatilization from sugarcane was investigated by Willis et al. (1969). Granular endrin (2% active ingredient) was applied by hand to a sugarcane plot located within a larger field of sugarcane at a rate of 2 lb/acre. Half of the endrin was applied to the soil surface and half to the sugarcane itself. A special apparatus, designed for continuous collection of volatilized endrin, was set up in the field. Air was drawn through the apparatus at a rate of 1 liter/min. This relatively low flow rate reduced the possibility of creating a "sink" which may have led to erroneous conclusions concerning the rate and extent of volatilization. The vapors were collected in an ethylene glycol trap, and the air was recycled back into the atmosphere. The collection apparatus, located 4 ft above the soil surface, was allowed to run continuously for 11 weeks following application of the pesticide. Ethylene glycol was replaced in the trap at three-day intervals thereafter. Thus each sample represented an average value for a given collection period. Analyses were performed by electron-capture gas chromatography, and the results are presented in Fig. 7.1. The cumulative recovery (in thousands of nanograms) is also given. The initial data point (540 ng/m^3) is an average concentration for the first three days following application and does not represent the highest level occurring during that time period. Shorter sampling periods would have been necessary in order to determine the actual peak concentration. The average atmospheric concentration decreased rapidly to 123 ng/m^3 at 21 days following treatment and continued to decrease less rapidly to a value of 30 ng/m^3 after 77 days. The asymptotic nature of the curve possibly indicates a dependence of volatilization upon concentration. The cumulative recovery approached 12,000 ng, and the shape of the recovery curve was also indicative of a decrease in rate of volatilization with time. It was estimated that about 5% of the total endrin applied was lost to the atmosphere; but this estimate was based upon assumptions as to the rate of air turnover above the plot and should not be taken as absolute.

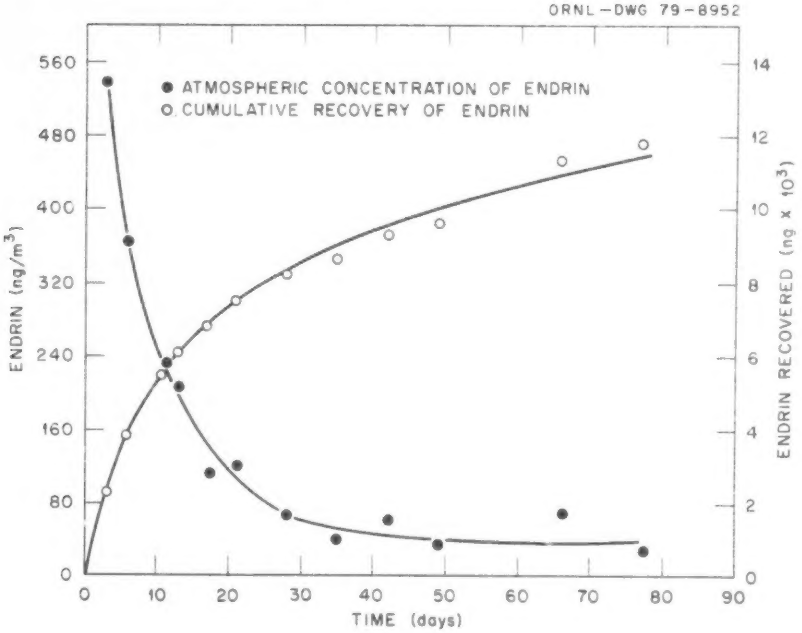

Fig. 7.1. Atmospheric concentration of endrin 4 ft above ground level in a sugarcane field and cumulative recovery. Source: modified from Willis et al., 1969.

A similar curve for disappearance of endrin from rice seedlings was obtained using an entirely different method (Ali and Nair, 1973). Rice seedlings were sprayed with an endrin emulsion, and plants were collected at given intervals following treatment. The concentration of endrin remaining in the plants was determined by exposing *Corcyra cephalonica* larvae to acetone extracts of the plants (which contain the residual endrin) and comparing the observed percent mortality to a standard dose-mortality curve. The results are presented in Fig. 7.2. While the primary factor in the loss of endrin activity was most likely volatilization, other possibilities, such as decomposition to compounds nontoxic to the test organisms, are not to be ruled out by this type of study.

The disappearance of endrin from treated cabbage was studied by Mattick et al. (1963). An emulsified formulation of endrin was sprayed on maturing cabbage at rates of 0.8, 0.5, and 0.25 lb of actual pesticide per acre. The cabbage was harvested on successive days following treatment and analyzed for endrin and its degradation products by electron-capture gas chromotography. The results are presented in Table 7.1. In each case, more than 95% of the endrin present in the cabbage at the time of treatment was lost within 21 days. Presumably, a fair percentage of this loss can be attributed to vaporization.

In another study (Klein and Korte, 1967), 1 to 2 mg ^{14}C-labeled endrin per plant was applied to white cabbage. Four weeks following application, only 6% of the radioactivity could be found in plants and

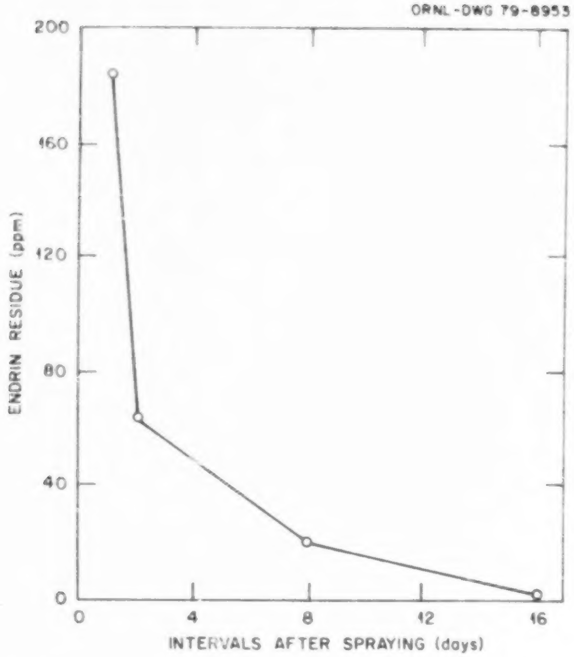

Fig. 7.2. Endrin residues remaining in rice plants as determined from mortality of *Corcyra cepholonica* larvae. Source: K. A. Ali and M. R. G. K. Nair, *Madras Agric. J.* 60(7): 439-440 (1973). Copyright 1973 Tamil Nadu Agricultural University.

Table 7.1. Disappearance of endrin residues from cabbage

Days after spraying	Endrin (ppm) remaining at each treatment level		
	0.8 lb/acre	0.5 lb/acre	0.25 lb/acre
0	4.17	2.26	Sample lost
1	4.79	2.45	0.72
2	2.36	1.06	0.50
5	2.08	0.48	0.34
7	0.81	0.32	0.21
10	0.95	0.18	0.10
14	0.30	0.17	0.09
21	0.13	0.10	0.004
% remaining (day 21)	3.1	4.4	0.56[a]
% lost (day 21)	96.9	95.6	99.44

[a] Based on day 1 value.

Source: Reprinted with permission from L. R. Mattick et al., *J. Agric. Food Chem.* 11(10): 54-55 (1963). Copyright 1963 American Chemical Society.

soil; the remaining 94% had disappeared into the atmosphere. The authors attributed 60% of the loss to evaporation of 34% to transpiration by the plants. This interpretation may also apply to the previous study as well.

The extent of pesticide vaporization from plants and soil under field conditions is determined by a complex, interacting collection of parameters and is not easily predicted. Some of these parameters, such as the vapor pressure of the pesticide, the temperature of the soil, and the wind velocity at the time of measurement, are obvious. Others are revealed only through extensive experimentation. While each observed parameter influences the volatilization of every organochlorine insecticide, the direction and extent of that influence may differ drastically among the various compounds. One particularly important variable appears to be the organic content of the soil in question (Bowman et al., 1965; Adams, 1967; Frost, 1969; Chesters and Konrad, 1971). Due to their lipophilic, hydrophobic nature, the chlorinated hydrocarbon insecticides (including endrin) strongly absorb to the organic material present in soil. Vaporization is therefore retarded from soils rich in organic matter. This phenomenon may also explain why the rates of vaporization are highest immediately following pesticide application, before the pesticide has become completely adsorbed.

The effect of soil organic matter on the vaporization of a series of chlorinated insecticides was studied by Bowman et al. (1965). Eight types of soil representing a wide range of organic content were equilibrated at 45°C to ensure roughly comparable moisture retention at the start of the experiment. Solutions of each pesticide in minimal amounts of hexane were added to samples of each of the soils, and the samples were incubated in a gravity convection-type oven at 45°C. Analyses for residual insecticide and degradation products were conducted after 4 and 8 days of incubation, using electron-capture gas chromatography. The results for dieldrin and endrin in three types of soil are presented in Table 7.2. Loss of dieldrin from Lynchburg loamy sand, poor in organic matter, was rapid and extensive. Following eight days of exposure, 88% had evaporated. Only 16% of the dieldrin was lost from Rutledge sand, rich in organic matter, after a similar incubation period. Endrin, however, did not behave at all like its isomer. Complete degradation to three products occurred in each soil type, and little loss was observed. Furthermore, the greatest relative loss (11%) took place from that soil containing the highest organic content. No explanation was presented for these unexpected results.

The moisture content of the soil also plays a role in determining the extent of volatilization of organochlorine insecticides (Bowman et al., 1965; Frost, 1969; Beall and Nash, 1971; Chesters and Konrad, 1971). In general, more volatilization is expected from wet soils than from dry soils. Bowman et al. (1965) studied the effects of moisture on the volatilization of a series of insecticides from four soils. Acetone solutions of each insecticide were applied to dry soil, to soil which was subsequently saturated with distilled water, and to soil previously saturated with distilled water. The treated samples were incubated at 45°C in a gravity convection-type oven for four days and analyzed for residues and degradation products by electron-capture gas chromatography. The results for dieldrin and endrin are presented in Table 7.3.

Table 7.2. Persistence and behavior of dieldrin and endrin in various types of soil at 45°C

Insecticide	Added insecticide recovered from soils after periods of exposure (%)			
	Days of exposure	Lynchburg loamy sand (0.17%)	Lakeland sand deep phase (0.42%)[a]	Rutledge sand I (6.56%)[a]
Dieldrin	4	24	50	93
	8	22	46	84
Endrin I[b]	4	0	0	0
II[c]	4	25	48	2
III[d]	4	17	0	33
IV[e]	4	56	45	54
Total endrin	4	98	93	89
Endrin I[b]	8	0	0	0
II[c]	8	39	50	1
III[d]	8	5	0	33
IV[e]	8	55	46	55
Total endrin	8	99	96	89

[a] Organic content in the soil (%).

[b] Calculated by comparison with endrin standard.

[c] Unknown degradation product of endrin.

[d] Endrin aldehyde.

[e] Endrin ketone.

Source: Reprinted with permission from M. C. Bowman, M. S. Schechter, and R. L. Carter, *J. Agric. Food Chem.* 13(4): 360-365 (1965). Copyright 1965 American Chemical Society.

In three of the four soil types, more dieldrin was lost from water-saturated soils than from dry soils. The exception was Rutledge sand. In all four soils, addition of dieldrin, prior to saturation with water, suppressed volatilization relative to the situation where saturation with water occurred first.

Loss of total endrin from water-saturated soils was greater than from dry soils in two of the four soil types. In this case the exceptions were Rutledge sand and Greenville clay. In three soil types, loss of total endrin from water-saturated soils was greatest when saturation occurred prior to addition of the insecticide. Here the exception was Lakeland sand. Once again, the complicated nature of the interactions involved renders predictions difficult.

A parameter closely associated with soil moisture content is humidity. Kalkat et al. (1961) studied the effects of relative humidity on the

Table 7.3. Effects of moisture on persistence and behavior of dieldrin and endrin in four types of soils exposed at 45°C for 4 days

Soil	Condition	Added insecticide recovered (%)					
		Dieldrin	Endrin				Total endrin
			I	II[b]	III[b]	IV[b]	
Lakeland sand deep phase (0.42%)	Dry soil	59	0	18	7	44	69
	H_2O + insecticide	43	15	8	4	31	58
	Insecticide + H_2O	48	21	8	4	33	56
Greenville sandy clay (0.57%)[a]	Dry soil	84	46	10	0	8	64
	H_2O + insecticide	61	40	2	0	8	50
	Insecticide + H_2O	67	67	2	0	8	77
Magnolia sandy loam (1.33%)[a]	Dry soil	83	6	40	0	33	79
	H_2O + insecticide	50	32	9	0	15	56
	Insecticide + H_2O	69	30	30	0	16	76
Rutledge sand II (19.43%)[a]	Dry soil	70	64	3	0	13	80
	H_2O + insecticide	75	72	1	0	11	84
	Insecticide + H_2O	84	92	3	0	2	97

[a] Organic content in soil (%).

[b] Degradation product of endrin.

Source: Reprinted with permission from M. C. Bowman, M. S. Schecter, and R. L. Carter, *J. Agric. Food Chem.* 13(4): 360-365 (1965). Copyright 1965 American Chemical Society.

volatility of several insecticides and found that their fumigant action increased significantly when the relative humidity was raised from 55 ± 5% to 90 ± 5% under similar temperature conditions (43.3°C in all cases). Loss of heptachlor epoxide through volatilization more than doubled when the relative humidity was raised from 7 ± 5% to 70 ± 5% at 34.4°C. The effect of increased humidity on the vaporization of several organochlorine insecticides, including endrin, was also studied (though unintentionally) by Bowman et al. (1965). In one case the insecticide solutions were added to soil previously dried at 45°C and returned immediately to a low-humidity environment. In the second case the insecticide solutions were added in an indentical manner, but the soil samples were returned to an oven also containing water-saturated soils and, therefore, to a highly humid atmosphere. The results for dieldrin and endrin in four soil samples after four days of incubation at 45°C are presented in Table 7.4. In three of the four soil types tested, the amount of dieldrin lost to the atmosphere was greater under humid conditions. Loss of total endrin was greater in all four soil types under humid conditions. Results in conflict with these have, however, also been reported. Lyon and Davidson (1965) studied the effects of humidity on the volatilization of certain insecticides. Insecticide residues were weighed before and after exposure in petri dish halves at 43.3 ± 1.1°C to a relative humidity of either 8 ± 5% or 80 ± 5%. The total weight loss for each group of replicate samples was determined. Of nine compounds tested, seven showed the expected greater weight loss at higher humidity. Two compounds, coumaphos and endrin, exhibited a greater weight loss at lower humidity. The total loss for endrin at 8 ± 5% relative humidity was 2.0 mg; at 80 ± 5% humidity, the loss was 1.1 mg. The exact cause of this devious behavior remains unknown.

Any treatment that increases the exchange of air over the soil surface will increase the rate of pesticide volatilization (Frost, 1969). The cultivation practice of disking or mixing the soil is an example of such treatment. Harvesting the crops also bares the soil to the air. Conversely, any treatment which reduces the air flow will retard volatilization. For example, pesticides persist much longer where a cover crop such as alfalfa is planted, or in forests where trees provide a canopy over the soil surface. In both of these instances, shade is also provided which helps keep the soil cool and retards vaporization (Frost, 1969).

A parameter which may possibly affect evaporation from crops is the surface area of the plant. Brett and Bowery (1958) studied the disappearance of endrin as a function of plant surface area. Three types of plants were used in these experiments: tomatoes (great volume with little surface area), collards (small volume with large surface area), and snap beans (intermediate in both surface area and volume). Crops were dusted in the field with 2% endrin and sampled at given intervals for analysis of residual pesticide levels; the results are presented in Table 7.5.

Collards, with the largest ratio of surface area to volume, contained the highest levels of endrin and retained residues for a longer period of time than did the other two types of vegetables. Endrin could not be detected in tomatoes one day following treatment.

Table 7.4. Effects of humidity on the persistence and behavior of dieldrin and endrin in four types of soils exposed at 45°C for 4 days

Soil	Condition	Added insecticide recovered (%)					Total endrin
		Dieldrin	Endrin I	II[b]	III[b]	IV[b]	
Lakeland sand deep phase (0.42%)[a]	Dry	50	0	48	0	45	93
	Humid	59	0	18	7	44	69
Greenville sandy clay (0.57%)[a]	Dry	94	0	49	0	34	83
	Humid	84	46	10	0	8	64
Magnolia sandy loam (1.33%)[a]	Dry	90	0	48	0	44	92
	Humid	83	6	40	0	33	79
Rutledge sand II (19.43%)[a]	Dry	97	47	5	5	41	98
	Humid	70	64	3	0	13	80

[a] Organic content in soil (%).

[b] Degradation product of endrin.

Source: Reprinted with permission from M. C. Bowman, M. S. Schecter, and R. L. Carter, *J. Agric. Food Chem.* 13(4): 360-365 (1965). Copyright 1965 American Chemical Society.

Table 7.5. Endrin residues on tomatoes, snap beans, and collards

Days after application	Tomatoes		Snap beans		Collards	
	Temp (°F max/min)	ppm	Temp (°F max/min)	ppm	Temp (°F max/min)	ppm
0	82/62	0.31	80/54	0.48	64/37	17.3
1	91/65	0[a]	81/58	0.25	64/36	15.2
2	96/71	0[a]	78/59	0.25	63/42	3.4
3	98/73	0[a]	81/62	0[a]	69/40	2.7
4	95/70	0[a]	85/59	0[a]	80/60	0[a]

[a] Below the minimum detection level (<0.10 ppm).

Source: C. H. Brett and T. G. Bowery, *J. Econ. Entomol.* 51(6): 818-821 (1958). Copyright 1958 Entomological Society of America.

While loss of residues may be attributed at least partially to volatilization, the role of surface area per se in determining the extent to which volatilization occurs might be questioned. The organic content of the plant surface would also be an important factor. Endrin may be more strongly adsorbed to the waxy cuticle of a collard leaf than to the surface of a tomato. This selective adsorption, in conjunction with a larger surface area, appears to provide a more complete explanation for the observed results.

Vaporization of endrin from soil surfaces may contribute to contamination of nearby plants. The extent to which vaporized endrin is taken up by the aerial portions of soybean plants was investigated by Beall and Nash (1971). Soybean seedlings were planted in specially designed pots which enabled the surface soil to contain 1.5 μCi of ^{14}C-labeled endrin at a concentration of 20 ppm, while the subsurface soil (to which the roots were exposed) remained pesticide free. Surface and subsurface soil layers were separated by a water- and vapor-tight disk as well as by an airspace. To confine vapors around the plant, a polyethylene cage, open at the top, was provided. The soil was kept moist for the duration of the 53-day experiment. At that time the plants were harvested and separated into lower stem, lower leaves, upper stem, upper leaves, pods, and seeds. Uptake of endrin was determined through complete combustion of the dried plant parts followed by quantification of the released ^{14}C-labeled CO_2. The results are presented in Table 7.6. Residue concentrations resulting from vapor sorption were highest in the leaves. The lower leaves contained more residues than the upper leaves, most likely

Table 7.6. Concentration of residues[a] found in soybean plants exposed to topsoil treated with ^{14}C-endrin

Plant part	Residues (ppm)
Upper leaves	15.95
Lower leaves	33.78
Total leaves	23.81
Upper stem	2.42
Lower stem	1.66
Total stem	1.89
Pods	2.84
Seeds	0.99
Average concentration[b]	6.7

[a] Based on ^{14}C content of combusted dried plant parts.

[b] Average concentration in total aerial parts.

Source: M. L. Beall, Jr. and R. G. Nash, *J. Environ. Qual.* 1(3): 283-288 (1972). Copyright 1972 American Chemical Society.

due to their closer proximity to the treated soil and their distribution over the soil. Pods contained higher concentrations than stems. The large hairy surfaces of the pods along with their semipendulous growth habits provided a greater surface area for vapor sorption than did the single upright stem. Although 43% of the applied endrin had vaporized by the end of the experiment, residue concentrations resulting from vapor sorption averaged only 6.7 ppm. One explanation is that most of the endrin may have volatilized during the early growth stages, at a time when the plants provided little canopy over the soil. Additionally, some of the residues originally adsorbed to the plant surfaces may have revolatilized and escaped to the environment. The authors suggest that the low organic content of the soil, the shallow (1-cm) depth of the treated soil layer, and the observance of moist conditions throughout the experiment may have resulted in a greater degree of volatilization than would occur in the field. These factors may, however, have been counterbalanced by the fact that the endrin was uniformly distributed throughout the soil as opposed to being applied merely to the surface.

7.3.2.2 Vaporization and codistillation from water. Vaporization and codistillation of organochlorine insecticides from fresh and marine waters is another well-established route of atmospheric contamination (Risebrough et al., 1968; Frost, 1969; Koeman et al., 1972). As a result of their tendency to accumulate at the surface of the water, these compounds are carried off into the atmosphere as the surface water evaporates. Most of the pesticides in laboratory suspensions are lost in this way within a few days (Frost, 1969), and the pesticide content of rivers and lakes subjected to surface runoff from agricultural lands is seen to decrease rapidly. No studies dealing with the extent to which endrin is lost from bodies of water have been reported. However, circumstantial evidence implies that such loss may be significant. Birds and fish were collected from Lake Nakuru in Kenya, and their tissues were analyzed for residues of organochlorine insecticides including endrin (Koeman et al., 1972). Residues in general were found to be extremely low, indicating that chlorinated hydrocarbon pesticides were not major pollutants at the time of sampling. Lake Nakuru, which has no outlet, is surrounded by agricultural land where insecticides (including endrin) are frequently used. Under the circumstances, it was expected that the residues in birds and fish would be equal to those found in similar ecosystems in other parts of the world. The results may indicate that the rate of disappearance of chlorinated hydrocarbon pesticides in tropical areas is relatively high as compared with more temperate areas and that vaporization and codistillation from water is a major route of disappearance. It is possible that tropical regions where pesticides are used extensively may be significant sources of the observed global atmospheric pollution by these compounds.

7.3.2.3 Other sources. Other potential sources of organochlorine insecticides in the atmosphere have not been studied specifically as they relate to endrin. These sources are, however, well established for similar compounds and therefore must be considered as probably routes of contamination for endrin as well. One major source of atmospheric contamination is pesticide-laden dust picked up directly from soils by winds

(Risebrough et al., 1968; Frost, 1969; Yobs et al., 1972). These airborne dusts are of more than local significance, since they may be transported thousands of miles by the prevailing winds, especially during storms. Pesticides adsorbed to large particulates (size range > 1 μ) will be deposited fairly close to their point of origin. Pesticides adsorbed to finer particles (size range < 1 μ) will travel greater distances. Risebrough et al. (1968) collected gram quantities of airborne particulate matter over Barbados. From the mineralogical and biological composition of these samples, it was apparent that they had originated from Europe and Africa and had traveled, carried by trade winds, over 6000 km across the Atlantic Ocean. Using the pesticide concentration on the dust (41 ppb) and estimates of the rate of dust sedimentation throughout the areas affected by the trade winds, Risebrough concluded that approximately 600 kg organochlorine insecticides was deposited over this portion of the Atlantic every year. This estimate represents a minimum figure, since the method of collection did not trap particles of less than 1 μ in diameter. The percentage of aerial dust-borne pesticides that actually arrive there as such is difficult to determine, since vaporization from, and/or adsorption to, particulates undoubtedly takes place in the atmosphere. It is possible that the dust itself did not represent the original source of the pesticides, but rather that free pesticide vapors, also carried by the winds, were picked up by dusts from totally different origins.

Another source of atmospheric contamination closely related to the one just discussed is aerial drift from spraying or dusting applications (Tabor, 1965; Abbott et al., 1966; Frost, 1969; Chesters and Konrad, 1971; U.S. EPA, 1972; Yobs et al., 1972). Such contamination is mainly local, with concentrations likely to vary inversely as the distance from the point of application. However, appreciable distances may be effected if the wind conditions are appropriate, and a potentially serious situation exists if a water body is located within drift distance. Ground spraying is four to five times less likely to result in drift than is aerial spraying, but aerial spraying is generally more popular. The best weather conditions for aerial spraying are on days when there is little or no wind and when a temperature inversion exists between 3 and 10 m above the ground (Chesters and Konrad, 1971). The temperature inversion prevents the upward movement of the pesticides and subsequent transportation by the wind. These ideal conditions seldom prevail, and significant amounts of pesticides are transported great distances following application. Due to their smaller particle size, dusting applications are more likely to result in widespread dissemination than are aerosolic applications. Some evidence for widespread drift of applied pesticides is the observation that the mineral talc used as a carrier and diluent for pesticides occurs in the solid-mineral phases of rain, glaciers, and rivers and in dusts recovered from the atmosphere in concentrations much higher than can be expected from natural occurrences (Risebrough et al. 1968). Unfortunately, this talc cannot be used as a quantitative tracer because it is gradually being displaced by water and light petroleum bases.

Vapors from industrial sources such as pesticide manufacturing or formulating plants are yet another source of atmospheric contamination (Tabor, 1965; Abbott et al., 1966). Once again, the greatest effects are local, but transport over great distances is possible.

One final route by which pesticides may be released to the atmosphere is during disposal of unused product or empty containers. The most popular method used by farmers to dispose of empty containers is burning in an open fire along with other trash. While burning containers is recommended by most manufacturers, reliable estimates suggest that temperatures obtained in open burning are not sufficient to destroy these compounds (U.S. EPA, 1972). As a result, vaporization occurs.

7.3.3 Monitoring for Endrin in the Atmosphere

Extensive monitoring for organochlorine pesticides in the atmosphere has not been conducted, and data for endrin are limited. One reason for the paucity of studies in this area is the difficulty of accurate analysis at low ambient levels. The most sensitive quantitative analytical technique available at this time is electron-capture gas chromotography. This technique, however, cannot distinguish among certain similar compounds, and the sought-after peaks are sometimes hidden by those of contaminants present in higher concentrations (such as the ubiquitous polychlorinated biphenyls). Mass spectrometry is an accurate qualitative tool. However, it is not very sensitive and cannot detect "trace" amounts of pesticide. Mass spectrometers are also very expensive and require highly trained technicians for their operation. As a result they are generally not available for confirmatory studies during ambient air sampling programs (Lewis, 1976).

Despite these problems, some studies have been reported. Tabor (1965) conducted a study in which the concentrations of particulate pesticide pollutants in the atmosphere of urban areas were measured. The communities chosen were surrounded by agricultural areas where large amounts of pesticides are applied annually during the growing season. If substantial amounts of pesticides were not found under these conditions, the probability of significant contamination in cities far removed from agricultural activity would be rather slight.

Sampling stations were set up in the center of town, and sampling was conducted during the periods of greatest application of pesticides to crops. Particulate matter was collected on glass-fiber filters and analyzed by electron-capture gas chromotography. The limits of detection were less than 1 ng (10^{-9} g).

Of the agricultural communities sampled, two had histories of endrin usage. Leland, Mississippi, and Newellton, Louisiana, are both surrounded by cotton fields where numerous pesticides, including endrin, are applied during the growing season. DDT and chlordane were detected at both sampling sites at maximum concentrations of 20 and 2 ng/m^3 respectively. Toxaphene and malathion were detected in Newellton. No trace of endrin was found in any samples from either community. These studies, however, were limited to particulate pesticide sampling. Accurate measurement of atmospheric pesticides requires a simultaneous quantitative collection of vapors as well as particulates. The concentrations reported here must be interpreted as minimum estimates, and it is quite possible that vaporized endrin is present in the atmospheres of both of these communities.

In 1970 and 1971, the Division of Pesticide Community Studies of the United States Environmental Protection Agency (no longer in operation) conducted a survey of pesticide levels in ambient air in the United States (U.S. EPA, 1970b; U.S. EPA 1971a; U.S. EPA 1971b; Yobs et al., 1972). During 1970, 30 sites in 19 states were sampled; during 1971, 45 sites in 27 states and the island of Bimini in the Bahamas were sampled. The sampling system consisted of a fiberglass cloth filter and an ethylene glycol trap through which air was drawn at a constant rate by a vacuum pump. Thus, both particulate matter and vapors were collected. Analysis was by electron-capture gas chromatography. Several problems inherent in this study became apparent at its conclusion (Lewis, 1976). Samples from stations located 5 miles apart had been pooled, even though one was representative of an urban community and the other of an agricultural area. Due to apparatus malfunction, airflow measurements were discovered to be inaccurate. Interfering compounds were present in the gas chromatograms, and confirmation of pesticide identity by mass spectrometry was not obtained. Therefore, both qualitative and quantitative estimates of ambient atmospheric concentrations, as determined by these studies, are generally unreliable. Nevertheless, these studies represent the only broad survey of ambient endrin concentrations, and the results are worthy of discussion. We can assume a 50% probability that the compound observed was, in fact, endrin. The chances are even less that the concentrations are correct (Lewis, 1976). The levels of endrin detected at those sites where it was observed are presented in Table 7.7.

Table 7.7. Endrin residues in ambient air 1970 and 1971

Site (by state)	Max endrin (ng/m^3)	Min endrin (ng/m^3)
	1970	
Alabama	0.9	0.6
Arkansas	8.8	0.3
Kentucky	3.1	1.5
South Dakota	1.8	0.5
Tennessee a	2.2	0.6
Tennessee b	2.2	0.4
	1971	
Arkansas	7.7	1.6
Colorado	25.6	0.2
Mississippi	0.9	*a*
Montana	2.9	*a*
Ohio	1.7	0.2
Oklahoma	4.6	0.3
Tennessee	12.4	0.2

[a] No minimum value reported.

Source: modified from U.S. EPA, 1970b, 1971a.

Endrin was detected in 6 out of 30 and 7 out of 45 sampling sites in 1970 and 1971 respectively. The maximum concentration reported in 1970 was 8.8 ng/m^3 in Little Rock, Arkansas; the maximum in 1971 was 25.6 ng/m^3 in Greeley, Colorado. Maximum levels for each year were reported during the pesticide use season as expected.

In another study (Stanley et al., 1971), air was sampled at nine locations in the United States and analyzed in a similar fashion for 19 pesticides and their metabolites. Sites were representative of both urban and agricultural areas, and levels were found to be much lower in the urban samples. Endrin was found at only one site, Stoneville, Mississippi, in 25 out of 98 samples collected. Stoneville is representative of large, southern agricultural areas. The highest concentration reported was 58.5 ng/m^3. Arthur et al. (1976) collected weekly air samples at Stoneville, using a similar technique, and found average yearly atmospheric levels of endrin (ng/m^3) as follows: for 1972, 3.2; for 1973, 2.3; and for 1974, 5.3.

In 1972 an eight-station experimental airborne pesticide monitoring network was established by the Syracuse University Research Corporation. Sampling was conducted over a 12-month period at each of the eight sites, using an experimental air sampling device. The device collected particulates on a fiberglass filter and adsorbed vapors onto cottonseed-oil-coated glass beads. Samples were analyzed for the presence of 14 pesticides by electron-capture gas chromatography. The limit of detection for endrin was 0.29 ng/m^3. No endrin was detected in any sample from any sampling site. Sites included six locations in upstate New York; Winter Haven, Florida; and Lubbock, Texas (Compton et al., 1972).

During this past year a small pilot program for monitoring pesticides in air was established by the National Human Monitoring Program for Pesticides of the United States Environmental Protection Agency. From April through July 1975, 15 air samples were collected and analyzed from suburban sites in Miami, Florida; Jackson, Mississippi; and Fort Collins, Colorado. Analysis was by electron-capture gas chromatography. Only one sample contained endrin at a concentration of 0.5 ng/m^3; this sample was collected at the Jackson, Mississippi, site (Kutz et al., 1976).

The final study to be reported was designed to measure the extent of air contamination in the homes of occupationally exposed men (Tessori and Spencer, 1971). Inside and outside air was monitored monthly for one year at the homes of 12 families in which the head of the household worked directly with pesticides. The collection device, installed for five days each month, consisted of a nylon chiffon screen saturated with ethylene glycol and suspended in a wooden frame. The screen trapped both particulates and vapors but was not designed to measure concentrations in any given volume of air. After each five-day sampling period, the screen was removed, extracted, and analyzed for pesticides by electron-capture gas chromatography. The results for two groups of exposed workers (five farmers and seven formulators) are presented in Table 7.8.

Table 7.8. Concentrations ($\mu g/m^2$) of endrin collected on ethylene-glycol-saturated screens

Statistic	Farmers (5)		Formulators (7)	
	Indoor air	Outdoor air	Indoor air	Outdoor air
N	38	37	52	54
$\underline{F}$	15	24	30	36
N$\underline{X}$	0.17	0.13	0.50	0.13
FX	0.43	0.19	0.86	0.20
Range	0.07-1.88	0.04-0.53	0.24-2.69	0.04-1.70

Note: N = number of samples taken,
$\underline{F}$ = frequency of endrin found out of N,
N$\underline{X}$ = numerical mean,
F$\underline{X}$ = frequency mean.

Source: J. D. Tessari and D. L. Spencer, *J. Assoc. Off. Anal. Chem.* 54(6): 1376-1382 (1971). Copyright 1971 Association of Official Analytical Chemists.

Outdoor air samples exhibited similar degrees of contamination for both groups, although the highest collected level outside of a formulator's home (1.70 $\mu g/m^2$) was over three times as high as the outside of a farmer's home (0.53 $\mu g/m^2$). Since the sample locations were widely distributed throughout the study area (Greeley, Colorado), it appears likely that these levels prevail throughout the entire area and are indicative of the ambient levels to which the general population is exposed. Analyses of indoor samples revealed that formulators bring home two to three times as much endrin on their person as do farmers. Consequently, the families of pesticide formulators are exposed to somewhat higher ambient concentrations than are either the families of farmers or the general population.

Although available data are far too limited to yield definite conclusions concerning the ambient levels of endrin in the atmosphere, certain trends do appear probable.

1. Endrin concentrations are highest in the atmosphere over agricultural areas and probably reach their peak levels during the pesticide use season.

2. Of all urban communities, those surrounded by farmlands run the highest risk of atmospheric contamination. Endrin adsorbed to particulates could not be detected in the air over representative communities, but may perhaps be present at very low concentrations in the vapor phase. Urban communities far removed from agricultural areas are unlikely to experience significant contamination.

3. The homes of occupationally exposed workers have higher levels of atmospheric contamination than do those of the general population. Formulators appear to bring home more endrin than do farmers.

4. In the few studies reported to date, no levels approaching the threshold limit value of 100 μg/m^3 adopted by the American Conference of Governmental Industrial Hygienists for 1971 have been detected (Yobs et al., 1972).

More extensive monitoring data, using confirmatory techniques such as mass spectrometry, are needed before further conclusions may be drawn concerning the concentration, distribution, and residence time of endrin in the atmosphere.

7.3.4 Mechanisms for Removal of Endrin from the Atmosphere

The majority of organochlorine insecticides released to the atmosphere are eventually returned to the earth's surface. Pesticides adsorbed to large, heavy particulates usually fall out close to their point of origin, while those associated with the smaller, lighter dusts or present as vapors may travel long distances before redoposition takes place. Cleansing of the atmosphere may occur as a result of particle fallout or washout of both particles and vapors during precipitation. Rainwater samples were collected continuously at seven widely distributed sites in the British Isles from August 1966 through July 1967 and analyzed for the presence of organochlorine insecticides (Tarrant and Tatton, 1968). Even though the sites were chosen so as to represent a variety of locations from urban to remote, the average total pesticide concentrations in annual rainfall were surprisingly similar (range, 104 to 229 ppt). Assuming an average pesticide concentration of 170 ppt in rainfall throughout the entire British Isles, it may be calculated that, in this area, 1 in. of rainfall would deposit 1 ton of pesticide. Since the average annual rainfall in the British Isles exceeds 40 in., approximately 40 tons of pesticide is deposited annually by this route. This value is four times greater than the quantity of pesticide annually dumped into the Gulf of Mexico by the Mississippi River (Frost, 1969). Endrin was not detected in any of the samples; however, endrin is not commonly applied in Britain, and there is every reason to expect that endrin would be detected in the rainfall of countries (such as the United States) which use this compound extensively.

The photochemical isomerization of endrin is a well-established phenomenon, and a variety of products, all considerably less toxic to mammals than is the parent compound, are formed (Klein and Korte, 1967; Soto and Deichmann, 1967; Plimmer, 1971).

Due to the difficulty of simulating atmospheric conditions in the laboratory, all studies have been conducted with endrin either in the solid state or dissolved in organic solvents. It is believed, however, that similar reactions occur in the vapor phase and when endrin is adsorbed to airborne particulates. A great deal of work is needed in the area of atmospheric photodecomposition in order to assess the extent to which this process contributes to the removal of endrin from the atmosphere. For a more in-depth discussion of endrin photodecomposition, refer to Sect. 2.3 and 7.5.

Other pathways for removal of endrin from the atmosphere include inhalation by animals and birds and absorption and metabolism by airborne microbes. Aside from the obvious undesirability of these mechanisms, their contribution to cleansing the air is, at best, minimal.

From the information available, it may be concluded that contamination of the atmosphere by endrin per se at any given moment is a transient occurrence, and that the major hazard involved in release of endrin to the atmosphere is the subsequent contamination of the hydrosphere and lithosphere. This situation results from the fact that the established mechanisms for removal of endrin from the atmosphere do not remove endrin from the environment, but rather relocate endrin to other environmental compartments. Some dissipation via photodecomposition is possible; however, no real evidence that such transformations actually take place is available.

7.4 ENDRIN IN THE HYDROSPHERE

7.4.1 Introduction

The pathways for the entrance of endrin into the hydrosphere are numerous. They include: surface runoff following application of endrin to soil and crops, runoff from endrin-coated seeds, contaminated effluents from pesticide manufacturing and formulating plants, careless aerial application, dumping of unused pesticide into waterways, cleaning spray equipment in rivers and lakes, and contaminated rainout and fallout. Some of these routes may be controlled by voluntary action or legislation. Others are inevitable consequences of application of the pesticide.

Considerable progress has been made in recent years in the development of suitable technology for assessing the extent of hydrospheric contamination by organochlorine insecticide. The older carbon adsorption sampling systems were not very accurate, since quantitative retention of pesticide was not guaranteed (Lauer et al., 1966). In addition, due to the limited sensitivity of the analytical methods available prior to the later 1960s, large sample volumes were required. With the development of electron-capture gas chromatographic techniques for organochlorine compounds, ppt quantities in water could be detected, and much smaller sampling volumes were required. At present, 1-gal or 1-qt grab samples are sufficient for accurate analyses, and extensive monitoring is easily achieved. When levels of contamination are expected to approach the limits of sensitivity, larger grab samples may be collected and concentrated to an appropriate volume. The state of the art is therefore far more advanced in the case of hydrospheric monitoring than in the atmospheric counterpart, where complicated sampling equipment must be installed individually at each site. The large collection of monitoring data available for pesticides in water testifies to this fact.

Several points must be considered when interpreting the monitoring data for endrin, for it is virtually insoluble in water (Pfister et al., 1969). The failure to detect endrin in filtered water samples is therefore not indicative of the absence of hydrospheric contamination. Endrin is carried in water as a suspension, as an emulsion, or, most commonly, adsorbed onto soil and sediment particles. Thus, suspended particulates as well as water must be analyzed. The old carbon adsorption methods filtered out particulates and therefore led to minimum estimates of contamination. With the new grab-sample techniques, both water and particulate matter are analyzed.

If both water and suspended particulate are endrin free, it is still not safe to conclude that the hydrosphere is free of the pesticide. Recent contamination may be ruled out; however, previous episodes of contamination will have left their more persistant mark on the bottom sediment. It is therefore important that bottom deposits also be monitored. While less of a threat to life, contamination of bottom deposits with endrin implies a chronic source of contamination throughout the hydrosphere.

In general, the worst effects of contamination in a given body of water are observed immediately following the episode of pollution. Due to such processes as codistillation and sedimentation, a large proportion of the endrin is either transferred somewhere else or rendered relatively harmless. However, entire populations of birds and fish may be destroyed during the short period of acute contamination, and years may be required for the ecosystem to return to normal (Breidenbach et al., 1967; Rowe et al., 1971; Anonymous, 1975c).

7.4.2 Sources of Endrin in the Hydrosphere and Sediment

7.4.2.1 Surface runoff following application. The major source of endrin in rivers and other freshwater bodies is surface runoff from fields and crops following application (U.S. EPA, 1972; Chesters and Konrad, 1971). The factors which affect the extent of surface runoff contamination are numerous and complex. Included among these are: the rate of application of the pesticide, the runoff volume, the amount of intensity of precipitation, irrigation practices, topographic relief, soil permeability, soil organic content, and the degree of vegetative cover. Where intense precipitation falls on soil of low permeability and low organic content (e.g., soils with high clay content), runoff usually occurs. Steep topography accentuates runoff losses, since soil erosion is more severe, water infiltration is reduced, and, as a result, movement of soil particles is increased (Working Group on Pesticides, 1970). Conditions opposite to these would be unfavorable for excess surface runoff, and loss of pesticide by this route would be comparatively small.

Studies were conducted in 1961 and 1964 to determine the modes, extent, and duration of surface water contamination by endrin from sugarcane fields near Franklin, Louisiana (Lauer et al., 1966). From the end of June through August 1961, the Bayou Yokely basin, containing 3300 acres of sugarcane, was treated with almost 2000 lb of endrin. A carbon adsorption unit was operated continuously at the downstream end of Bayou Yokely from late April through November 1961. Of 18 samples collected by this unit, six contained endrin at concentrations ranging from 1 to 360 ppt, with a median concentration of 100 ppt. The occurrence of these residues in relation to endrin usage, rainfall, and runoff is shown in Fig. 7.3. Detectable (>1 ppt) quantities of endrin were recovered from Bayou Yokely water during the 90-day period July 8 to October 5, 1961, commencing shortly after the first of three sets of endrin applications in the basin and extending until shortly after the final set. The first and third sets, which totaled 500 and 800 lb of technical endrin, respectively, were followed by high levels of endrin recovery from the stream. Frequent intense rains and runoff occurred throughout both of these sampling periods.

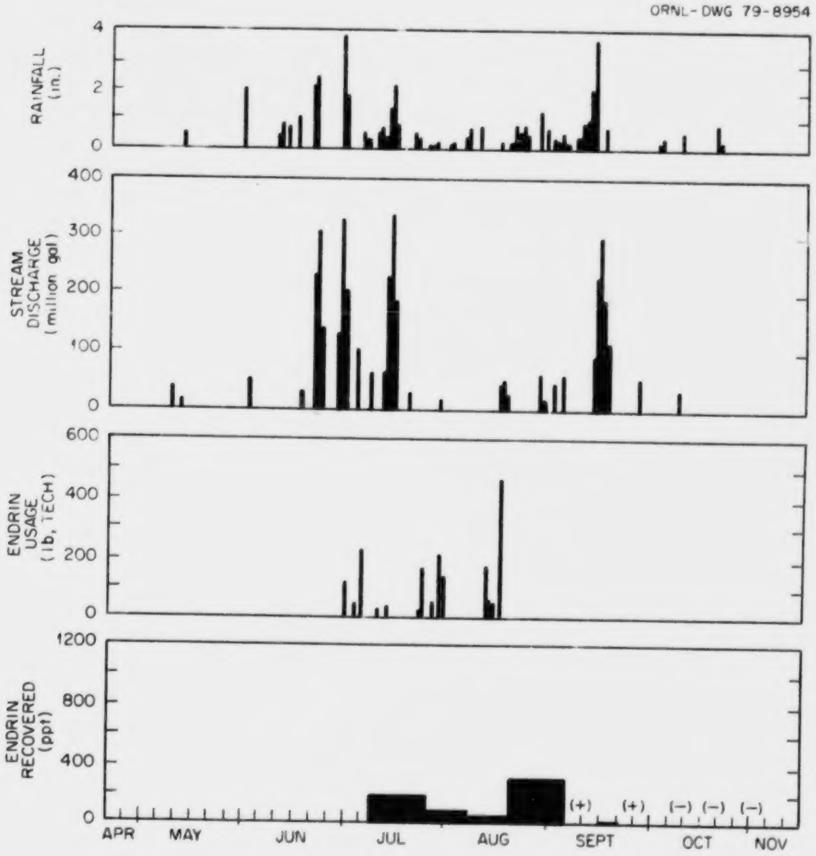

Fig. 7.3. Results from endrin pollution study of the Bayou Yokely watershed in Louisiana during 1961. Lower panel: pesticide recoveries from Bayou Yokely water, (+) sampled; endrin recovery at level of 1 ppt, (-) sampled; endrin, if present, was recovered at less than 1 ppt. Source: G. J. Lauer et al., *Trans. Am. Fish. Soc.* 95(3): 310-316 (1966). Copyright 1966 American Fisheries Society.

Six hundred pounds of endrin was applied during the second set of treatments. In this case, however, recoveries decreased during two subsequent sampling periods. Few intense rains and little runoff occurred during these sampling periods.

In 1964, both carbon adsorption samples and 1-gal grab samples were collected from each of three stations along Bayou Yokely at given intervals following the endrin application season. The maximum recoveries from grab samples and carbon adsorption samples taken from the second to the fourth of September were 820 and 700 ppt respectively. The mean recoveries from the three stations on those dates were 440 ppt for grab samples and 530 ppt for carbon adsorption samples. Recoveries of endrin still averaged 30 and 40 ppt for the two methods three months following the final application to the fields. Although less endrin (1200 lb) was used in the basin in 1964 than in 1961, larger recoveries over a longer period of time were observed. These results probably reflect both the improved sampling methods available at the time of the later study and the occurrence of more rain during October and November 1964 as compared with 1961. However, the pattern of surface water contamination remained the same in both studies. Maximum recoveries of endrin were observed immediately after the initial rains and runoff following application; but recoveries rapidly decreased thereafter.

Sediment and soil samples were also collected during the 1964 study. Endrin recovery from sediment was 165 ppb immediately following the application season in early September. Although concentrations decreased significantly during the subsequent three-month period, samples still contained an average of 70 ppb in late November, when the study was discontinued. Difficulties in obtaining representative samples of soil resulted in highly variable values for endrin contamination. However, concentrations averaging 1450 ppb were detected in soil at the conclusion of the study. The persistence of endrin in soil over a period of months implies that contamination of surface waters as a result of runoff probably takes place long after the application season is over.

Several studies have been conducted which deal specifically with the relationship between rainfall and runoff contamination. In July 1967 and 1968, endrin was surface-applied to sugarcane plots in Louisiana at the rate of 0.337 kg/ha, and surface runoff was sampled following each subsequent precipitation event (Willis and Hamilton, 1973). Rainfall, runoff volume, runoff contamination, and endrin loss data are presented in Table 7.9; concentrations remaining in soil are presented in Table 7.10. In 1967 the amount of rainfall varied considerably, ranging from 0.53 to 11.43 cm. Runoff volumes varied with the amount of rainfall but were also influenced by other factors such as rainfall intensity and soil surface conditions. The highest endrin concentration (1.23 ppb) occurred with the first runoff event three days following application. Since the weather conditions remained uncooperative during the immediate posttreatment period, a sprinkler system was employed to simulate rainfall on the first two sampling dates. It is believed that had the runoff occurring on day 3 resulted from 2.54 cm of natural rainfall, the intensity and impact energy would have been greater, and endrin loss in runoff would have been higher.

Table 7.9. Rainfall, runoff, and endrin loss in runoff from sugarcane plots following a single endrin application

Date of runoff	Rainfall (cm)	Runoff volume (m^3/plot)	Conc. in runoff[a] (ppb)	Lost (mg/ha)
		Endrin applied on July 31, 1967		
August 2, 1967	2.54[b]	0.24	0.37–2.73 1.23	66.05
August 14, 1967	3.18[b]	0.48	0.20–1.34 0.53	57.0
August 27, 1967	11.43	1.40	0.13–0.53 0.30	94.49
September 11, 1967	1.27	0.17	0.10–1.03 0.40	15.28
September 12, 1967	2.21	0.27	0.13–0.51 0.30	18.25
September 27, 1967	0.53	0.03	0.09–1.12 0.53	3.11
December 10, 1967	7.09	1.39	0.07–0.18 0.14	43.68
December 27, 1967	2.01	0.24	0.10–0.44 0.19	10.09
January 2, 1968	4.19	0.64	0.06–0.42 0.21	30.02
January 9, 1968	0.81	0.09	0.04–0.19 0.11	2.34
February 19, 1968	2.39	0.08	0.12–0.25 0.20	3.65
March 4, 1968	1.50	0.16	0.08–0.27 0.17	5.97
March 22, 1968	4.52	0.44	0.01–0.12 0.06	5.94
May 17, 1968	3.63	0.31	0.00	
		Endrin applied on July 13, 1968		
July 19, 1968	5.08	0.54	0.94–5.02 2.85	346.8
July 22, 1968	3.51	0.15	0.63–3.00 1.95	65.5

Table 7.9 (continued)

Date of runoff	Rainfall (cm)	Runoff volume (m^3/plot)	Conc. in runoff[a] (ppb)	Lost (mg/ha)
August 5, 1968	3.15	0.06	0.15–2.88 1.19	17.0
August 13, 1968	9.32	0.57	0.75–4.09 1.56	199.5

[a] Data from six plots. The first two numbers refer to the minimum and maximum concentration; the lower number is the mean concentration.

[b] Sprinkler irrigation used to simulate rainfall.

Source: G. H. Willis and R. A. Hamilton, *J. Environ. Qual.* 2(4): 463-466 (1973). Copyright 1973 American Society of Agronomy.

Table 7.10. Concentration of endrin in soil

Date		Concentration (ppb) at depth of	
Application	Sample	0–15 cm	15–30 cm
July 31, 1967	January 10, 1968	7.5–9.0 8.1	0.0
	July 9, 1968	1.2–6.5 3.3	0.6–1.2 0.8
July 13, 1968	July 29, 1968	34.2–124.9 76.3	2.1–13.0 5.2
	August 22, 1968	17.4–51.8 42.9	2.2–5.0 3.4

[a] Data from six plots. The first two numbers refer to the minimum and maximum concentration; the lower number is the mean concentration.

Source: G. H. Willis and R. A. Hamilton, *J. Environ. Qual.* 2(4): 463-466 (1973). Copyright 1973 American Society of Agronomy.

Endrin concentrations decreased to 0.53 ppb or less during subsequent runoff events, and no endrin (<0.01 ppb) was detectable ten months after application.

Endrin losses were greatest in the first month following application, although a significant loss occurred in mid-December during a storm that resulted in a large volume of runoff. Accumulated losses in runoff during the ten months following a single application amounted to only 0.1% of that applied.

Only four runoff events occurred between the endrin application in July 1968 and harvest in November. Endrin concentrations ranged from 1.19 to 2.85 ppb, with an accumulated loss of 0.2% of that applied. As in 1967, runoff contamination was highest immediately after application and decreased with time. However, a large runoff event occurring one month after treatment resulted in considerable loss.

In 1969, as part of the same study, Willis and Hamilton investigated the effect of the time interval between application and runoff on the amount of endrin found in the initial runoff and soil. A sprinkler irrigation system was used once again to simulate rainfall and initiate runoff 24 or 72 hr after application. The results are presented in Table 7.11. Mean concentrations of endrin in runoff water when rainfall occurred 24 hr after application were more than twice those observed when rainfall was postponed for 72 hr. The lower concentrations after 72 hr may have been due to the smaller amounts of endrin remaining at the soil surface as a result of volatilization. The lower endrin concentrations associated with the 72-hr time interval between application and rainfall were also reflected in soil concentrations 5.5 months following application. Approximately 43 and 17% of the applied endrin were present in soil receiving rainfall after 24 and 72 hr respectively.

Table 7.11. Effect of time interval between endrin application and rainfall[a] on endrin concentration in runoff and soil[b]

Time between application and rainfall (hr)	Concentration (ppb)	
	Runoff[c]	Soil
24	0.00–2.12 1.06	39.0–83.4 64.9
72	0.07–0.84 0.46	11.3–55.9 24.8

[a] Sprinkler irrigation used to simulate rainfall.

[b] Soil samples (0 to 15 cm) collected 5.5 months after application.

[c] Data from six plots. The first two numbers refer to the minimum and maximum concentration; the lower number is the mean concentration.

Source: G. H. Willis and R. A. Hamilton, *J. Environ. Qual.* 2(4): 463-466 (1973). Copyright 1973 American Society of Agronomy.

The effect of rainfall upon endrin contamination of the surface waters of Greenville, Mississippi, was studied by Iverson (1967). The study area consisted of 300 acres of cultivated land and contained a slough in which the surface runoff from the fields was collected. During 1965, when the study was conducted, a cotten field within the 300-acre tract received weekly applications of endrin from July 14 through September 3. Water from the slough was sampled on 19 different occasions between April 1965 and February 1966, and samples of runoff water were obtained whenever precipitation caused water to move rapidly and in quantity from the treated areas. The dates on which endrin was detected in the slough or runoff and the concentrations present on those dates are summarized in Table 7.12.

Table 7.12. Concentration of endrin in a drainage slough in Greenville, Mississippi, and in surface runoff

Slough		Runoff	
Date	ppb	Date	ppb
Apr. 20	0.18	May 22	0.96
May 17	0.72	May 27	1.37
Aug. 9	0.09	Sept. 9	0.12
Aug. 23	0.72	Sept. 21	0.21
Sept. 8	0.07	Jan. Feb. [a]	0.72 1.74 3.34
Feb. 21	1.49		

[a] No exact dates given.

Source: L. G. K. Iverson, *Agriculture and the Quality of Our Environment* (1967). Copyright 1967 American Association for the Advancement of Science.

Measurable residues of endrin were detected on only six of the sampling dates. The highest level (1.49 ppb) occurred in late February, during which time, heavy rains caused flooding in the study area, and at the peak residue period, 6 in. fell within 11 hr. Samples of runoff contained up to 3.34 ppb endrin.

The surface water contamination resulting from irrigation and rainfall following foliar application of endrin to cotton was studied by Sparr et al. (1966). A 5-acre cotton field in Kelso, Arkansas, was sprayed with 2 lb of endrin on three successive occasions. Irrigation water flowed through a furrow within the plot to a drainage ditch, which was sampled as it left the field. The ditch ended in a check dam, where the water was held for reuse. It was estimated that the water was diluted 20-fold at the dam site. During irrigation, water samples were collected before and after spraying from the irrigation furrow and the drainage ditch at the check dam. Rain runoff samples were taken from the end of a furrow during a rain storm. The results of analysis for endrin are presented in Table 7.13.

Table 7.13. Residues in water from spraying a cotton field with 0.4 lb/acre endrin

	No. of sprays	Interval from last spray (days)	Endrin conc (ppb)
Input irrigation H_2O	1	1	0.08
Used irrigation H_2O from furrows[a]	1	1	0.36
Input irrigation H_2O	2	3	0.12
Used irrigation H_2O from furrows[a]	2	3	0.11
Rain runoff, 1.15 in.	3	40	0.05
	1	7	0.66
H_2O held by check dam 5/8 mile from plot	1	2	0.006
	1	3	0.008

[a] 1/4 mile long.

Source: B. I. Sparr et al., *Agriculture and the Quality of Our Environment* (1967). Copyright 1967 American Association for the Advancement of Science.

A 4.5-fold increase in endrin concentration (0.08 ppb to 0.36 ppb) was observed in the irrigation water when sampled one day following the initial application. About five-sixths of the endrin in this sample (0.30 ppb) remained on a glass filter, indicating that it was adsorbed to soil particles. Three days after the second application, the input irrigation water contained 0.12 ppb endrin. No increase in concentration was observed following irrigation on the same day. In this case, however, only one-tenth of the endrin was retained on a filter. It may be concluded that more soil was dislodged during the first irrigation than during the second, and that contamination of surface runoff is dependent to a large extent upon the degree of soil errosion. Forty days after the final spraying, the endrin concentration in the used irrigation water was only 0.05 ppb. The concentration of endrin in water held by the check dam was less than 10 ppt, significantly less than could be accounted for by the 20-fold dilution. No endrin was detected in the mud from the check dam, although the analytical sensitivity was only 0.01 ppm. Whether the observed 45-fold dilution was due to adsorption onto sediment or to loss by evaporation is not certain. However, these concentrations are believed to be representative of those which may enter streams from cotton fields contiguous to them.

A 1.15-in. rainfall which occurred seven days after the first application gave rise to the highest water concentration observed in these studies (0.66 ppb). This high concentration may have resulted from the washing of endrin off the foliage, which probably held most of the applied pesticide. This conclusion was supported by soil residue data, which indicated that only 10% or less of the applied endrin (0.04 to 0.02 ppb) was lost from the foliar canopy at the time of application. If all of the endrin had reached the ground, the concentration in soil would have been 0.4 ppb.

In June 1963, the Cedar Bluff Irrigation District was created along the Smokey Hill River from Cedar Bluff Dam eastward in central Kansas. This area was transformed from one of dry-land farming practices with little insecticide usage to land with intensified crop production requiring the use of significant quantities of pesticides. The extent of surface-water and groundwater contamination by endrin as a result of this transformation was investigated in a four-year study conducted from 1965 through 1969 (Knutson et al., 1971). A cornfield of approximately 3.25 acres was sprayed with 0.3 to 0.4 lb/acre endrin annually for the duration of the study. Foliage samples were collected soon after application and at harvest time along with grain samples. Water and silt samples were taken on the Smokey Hill River just above and below where drainage from the experimental field occurred, in the Cedar Bluff Reservoir near the dam, and where water entered the test field from the irrigation canal. Groundwater samples and samples of surface runoff were also collected. Foliage samples collected soon after spraying showed endrin residues of from 0.9 to 6 ppm over the four-year period. Endrin residues persisted in the foliage at harvest time (range, 0.06 to 2.43 ppm); however, none were detected in the corn grain. No residues were detected in surface runoff or groundwater at any time, or in reservoir and river silt or water in 1969. From 1966 through 1968, endrin was detected once in reservoir water at 13 ppt and twice in river water at 3 and 10 ppt. The absence of rainfall probably accounted for the persistance of large quantities of endrin on the foliage with little transference to the soil. Thus, the surface waters remained relatively free of pesticide.

Examples of extensive contamination of surface waters from pesticide-laden irrigation runoff are also available. One such example is the situation existing at the Tule Lake and Lower Klamath Lake National Wildlife Refuge in northern California during the early 1960s. The deaths of unusually large numbers of fish-eating birds were attributed to the use of pesticides on agricultural lands within the surrounding refuge. Drainage from these lands carried the pesticides through the extensive irrigation system serving the refuge. This situation prompted an investigation of the occurrence of chlorinated hydrocarbon insecticides at various locations within the Lost River system draining into and out of the refuge (Godsil and Johnson, 1968). Monitoring of water, suspended material, and various organisms (both free and caged) was conducted from April 1965 through January 1967. Endrin predominated in all analytical results due to its abundant usage (14,000 lb in 1966) and persistence in drainage water following application. Data for endrin contamination were reported for a representative location known as drainage pump B, which discharged into the Tule Lake sump. Water used throughout the entire irrigation system eventually passed through pump B, and model studies indicated that this water could be recycled in irrigated lands up to 5.2 times. Consequently, the highest levels of endrin relative to those found at other stations were observed there. The occurrence of endrin in the water and biota of the Tule Lake sump at pump B is shown in Fig. 7.4, and the data for water and organism analyses are presented in Table 7 14. Most striking is the increase and subsequent decrease of endrin in the water and all levels of biota during the main growing season from May through September.

During the two years shown (1965 and 1966), concentrations in water increased from below the levels of detection in April to peaks of 0.1 and 0.069 ppb, respectively, at the height of the growing season. The patterns of endrin contamination in captive fish paralleled those in water, with maximum accumulations of 97 ppb in 1965 and 107 ppb in 1966. Concentrations decreased once again to below the levels of detection in late fall and remained so throughout the winter. The most important conclusion is that short-term contamination of an aquatic environment with endrin does not result in permanent residual concentrations in water or biota. However, sediment samples were not analyzed in this study, and accumulation of pesticides in sediment, and their consequent low-level but continuous release, is certainly possible.

7.4.2.2 Runoff from endrin-coated seeds. Endrin is often applied to seeds prior to seeding operations in order to protect them from decimation by rodents. Loss of endrin from these seeds during runoff following precipitation or irrigation may result in contamination of neighboring streams. Samples of Douglas fir seed, coated with 1 lb of active endrin per 100 lb of seed and soaked in distilled water for varying lengths of time, rapidly lost endrin during the first hour (Marston et al., 1969). For the remainder of the 32-day experiment, endrin continued to be removed at a fairly constant but greatly reduced rate. The

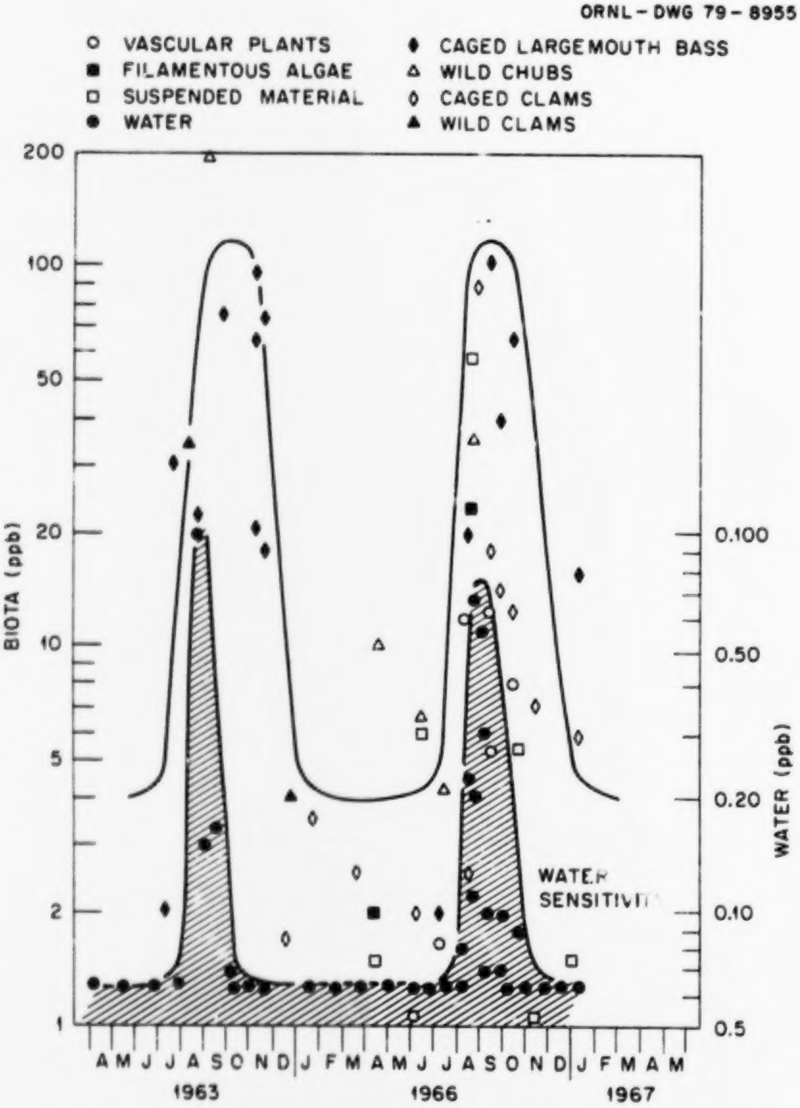

Fig. 7.4. Occurrence of endrin in water and biota at pump B. Source: Godsil and Johnson, 1968.

actual losses for a typical sample are presented in Table 7.15 and in Fig. 7.5, where a linear regression curve is also shown. The deviations from linearity observed in Fig. 7.5 were probably due to irregularities in seed shape and surface area and to differences in the thickness of the endrin coating. The total amount of endrin recovered, 28.2 ppb, amounted to approximately 11.3% of the amount contained in the sample. In a field experiment using the same seed, losses were less pronounced. Grab samples of water from Needle Branch were collected and analyzed for endrin before, during, and after the aerial application of endrin-coated

Table 7.14. Pumb B water and biota analyses (residues) (ppb)

Date collected	Water	Date collected	Suspended material
4/01/65		4/20/66	1.5
5/14/65		6/22/66	6.0
6/22/65		7/22/66	1.3
7/22/65		8/22/66	57.7
8/11/65	0.100	9/13/66	13.0
8/27/65	0.015	10/26/66	5.3
9/10/65	0.017	11/16/66	
10/01/65	0.007	1/06/67	1.5
10/11/65			
10/20/65			Vascular plants
11/09/65			
1/18/66			
2/17/66		6/22/66	
3/22/66		7/22/66	1.6
4/26/66		8/22/66	12.2
6/07/66		9/13/66	12.5
6/22/66		9/29/66	4.8
7/06/66		10/20/66	8.0
7/20/66		11/16/66	1.8
7/28/66			
8/04/66			Algae
8/05/66			
8/09/66	0.008	4/20/66	2.0
8/11/66		6/22/66	
8/18/66	0.023	7/22/66	
8/20/66	0.011	8/22/66	22.3
8/22/66	0.021	9/13/66	10.8
8/24/66	0.069		
8/26/66	0.057		Chubs
8/29/66	0.056		
9/02/66	0.030	8/27/65	198.0
9/07/66	0.007	4/20/66	10.0
9/13/66	0.010	6/22/66	6.0
9/22/66	0.007	7/22/66	4.0
9/29/66	0.010	8/22/66	30.5
10/04/66			
10/19/66	0.009		Clams
11/04/66			
11/18/66		8/10/65	34.0
11/25/66	0.007	12/28/65	4.0
11/30/66		7/22/66	2.0
12/14/66			
1/11/67			
2/06/67			

Table 7.15. Concentrations of endrin in water from samples of Douglas fir seed soaked for different lengths of time

Solvent	Length of soaking period (days)	Concentration (ppb)
Distilled water	1/24	1.34
	1/8	2.36
	1/4	3.65
	1	5.02
	1	2.98
	2	5.22
	3	10.2
	7	4.57
	7	7.26
	14	18.4
	21	20.0
	30	23.2
	32	28.2
pH 4.5–5.0	7	10.1
pH 4.5–5.1	7	10.5
pH 9.5–7.0	7	9.47
pH 9.5–7.0	7	11.2

Source: modified from Marston et al., 1969.

seeds to the Needle Branch watershed. Sampling started 0.5 hr prior to seeding on January 23, 1967, and continued at increasingly longer intervals through January 28. Three samples were also taken during a winter frost freshet that occurred on January 29; the results are presented in Table 7.16. The total amount of endrin detected in runoff amounted to 1.05×10^{-3} kg/km^2 or only 0.125% of that theoretically applied (0.84 kg/km^2). Ninety-six percent of this amount was lost during the winter freshet. The remaining 4% was lost during the 2-hr period after seeding started and probably came from loose powder that rubbed off the seeds.

In another study (Mann et al., 1971), two lots each of loblolly and slash pine seeds were coated with an endrin formulation and surface-sown. One lot of each type of seed had been presoaked for 24 hr prior to treatment in order to approximate the moisture content obtained by cold stratification, a procedure frequently employed to condition the seed.

Samples were taken from each sublot and analyzed for endrin content after 0, 15, 30, and 45 days of exposure to natural conditions; the results are presented in Table 7.17. After the first 15 days, more than 80% remained on three of the four lots. The soaked slash pine had apparently

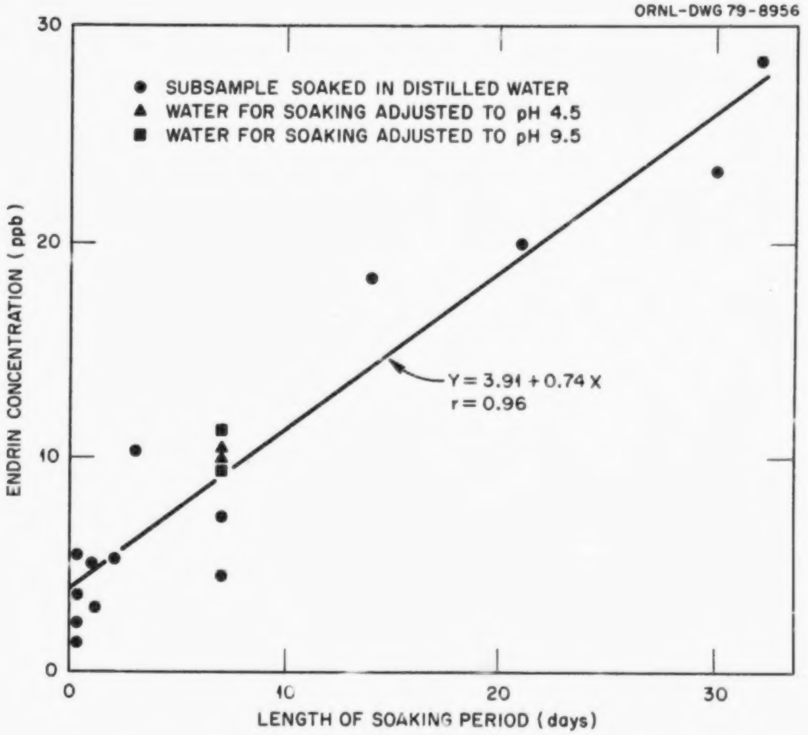

Fig. 7.5. Concentrations of endrin in water from subsamples of Douglas fir seed soaked for different lengths of time. Source: modified from Marston et al., 1969.

been coated too heavily (23.6 mg as opposed to an average of 12.6 for the other three lots) and, due to excess flaking, contained only 58% on the same sampling date. The small initial losses were attributed to low rainfall (only 0.16 in. total during that period). After 30 days the three lots treated correctly contained 60 to 70% of their endrin and had lost slightly more during this period than during the first 15 days.

Rainfall had been near normal, totaling 2.33 in. Between 30 and 45 days, above average rainfall occurred (3.36 in.) and resulted in heavy losses from all four lots. Only 22 to 23% of the endrin remained regardless of how much had originally been applied. Both studies make obvious the importance of rainfall in determining the extent of endrin loss from treated seeds.

7.4.2.3 Contaminated industrial effluents. Another route of endrin into fresh waters, of significance in certain localities, is the release of contaminated effluents from pesticide manufacturing or formulating plants (Tabor, 1965; Abbott et al., 1966; Barthel et al., 1969; Eichelberger and Lichtenberg, 1971). Studies of pesticide residues in sediments of the lower Mississippi and its tributaries conducted in 1964 and again in 1966 revealed one major area of endrin contamination — that associated with a

Table 7.16. Endrin analysis of water samples, Needle Branch Watershed, January 23-29, 1967

Sampling time	Instantaneous concentration (µg/liter)	Average concentration (µg/liter)	Streamflow (liters per square mile)	Weight of endrin	
				µg per square mile	lb per square mile
1/23/67					
2:30 PM	0	0			
3:00	0	0			
3:15	0.04	0.02	340,882	6,818	0.00002
3:30	0.10	0.07	340,882	23,862	0.00005
3:45	0.04	0.07	340,882	23,862	0.00005
4:00	0.04	0.04	340,882	13,635	0.00003
4:15	0.04	0.04	340,882	13,635	0.00003
4:30	0.04	0.04	334,812	13,392	0.00003
4:45	0.04	0.04	328,741	13,150	0.00003
5:00	0[a]	0.02	328,741	6,575	0.00001
1/29/67					
12:15 AM	0[a]	0			
10:30 AM	0.04	0.02	51,271,481	1,025,430	0.00226
11:15 PM	0	0.02	76,446,331	1,528,927	0.00337
Total					0.00588

[a] That no results are given for the samples taken between the one at 5 PM on January 23, 1967, and the one at 12:15 AM on January 29, 1967, indicates that no endrin was detected in any of the samples taken between these two 0 entries.

Source: Marston et al., 1969.

Table 7.17. Endrin remaining on 200 repellent-coated seeds after different periods of exposure

Days of exposure	Loblolly				Slash			
	Dry		Soaked		Dry		Soaked	
	(mg)	(%)	(mg)	(%)	(mg)	(%)	(mg)	(%)
0	10.5		10.3		17.1		23.6	
15	8.8	83.8	9.3	90.3	13.8	80.7	13.7	58.1
30	7.0	67.7	7.2	69.9	10.2	59.7	9.6	40.7
45	3.0	28.6	4.0	38.8	3.8	22.2	6.1	25.8

Source: Mann et al., 1971.

pesticide manufacturing operation in the Memphis Wolf River–Cypress Creek complex (Barthel et al., 1969). No endrin was found in bottom sediments from any of the other Mississippi River tributaries or from any of the 11 Mississippi River sampling sites investigated. In 1964, endrin was found in Cypress Creek at concentrations of 824, 12,800, and 172 ppm in sediment samples taken upstream, midstream, and downstream, respectively, from a primary manufacturer of endrin and heptachlor; in 1966, 10,200 ppm endrin was detected on a spoil bank 20 to 60 ft southwest of that stream. Old Wolf River was not sampled in 1964, but concentrations of 0.57, 0.26, and 0.31 ppm endrin in sediment samples taken upstream, midstream, and downstream, respectively, from the confluence with Cypress Creek were detected in 1966. A further study of endrin contamination of the Wolf River–Cypress Creek complex was conducted in April 1967. In this study, water as well as sediment samples was collected both upstream and downstream from (1) the pesticide manufacturing plant and (2) the confluence of Cypress Creek and Wolf River. Endrin was not detected in water from Wolf Creek but was present at a concentration of 1.01 ppm in a bank deposit sample from that stream. Water from Cypress Creek contained from 0.27 to 2.03 ppb endrin and 5.04 to 6.50 ppb endrin ketone. Concentrations of endrin in Cypress Creek sediment ranged from 0.24 to 23.6 ppm, and endrin ketone was detected at concentrations of 2.08 to 2.80 ppm. Samples of Cypress Creek dredge spoils contained from 47.4 to 10,676 ppm endrin and from 24.8 to 3509 ppm endrin ketone, while sediment from a stream deposition area contained 1.93 to 294 ppm endrin and 4.78 to 96.10 ppm endrin ketone.

Pesticide residues in Wolf River sediment samples upstream from the confluence with Cypress Creek probably originated from three sources: (1) Cypress Creek water from the vicinity of the manufacturing plant, transported upstream by backwater from the Mississippi River; (2) direct discharge of manufacturing wastes into the Wolf River or into the Mississippi River with subsequent transport upstream during high stages of the Mississippi River; and (3) seepage from wastes buried in recent years at a nearby dump.

7.4.2.4 Careless practices. Carelessness in the application and handling of pesticides represents yet another route of hydrospheric contamination, and specific cases of undesirable practices resulting in contamination by endrin have been reported (Lauer et al., 1966; Iverson, 1967; Saunders, 1969). In one study (Lauer et al., 1966) in which pilots were cautioned to avoid direct contamination of nearby streams during aerial spraying (a common source of pesticides in streams bounded by agricultural lands), a pilot was observed applying endrin immediately after a heavy rain, when water was running out of the fields. This incident undoubtedly contributed to the high concentrations observed in the drainage basin following application.

Practices such as filling pesticide sprayers at streams, washing spray equipment in streams, or draining excess spray materials into drainage facilities have been routinely observed. Saunders (1969) reported a case in which extensive mortalities occurred among brook trout and juvenile Atlantic salmon following the cleansing of an endrin-containing potato sprayer into Mill River, Prince Edward Island. Concentrations in the water at the time of the fish kill were not determined. The burning of insecticide containers, such as paper cartons or sacks, where ashes can blow or be washed into a stream must also be included among practices which lead to contamination, although no instance involving endrin per se has been reported.

7.4.2.5 Contaminated fallout and precipitation. A detailed discussion of the pathways by which endrin enters the atmosphere was presented in Sect. 7.2.2. Loss to the atmosphere does not, however, represent a mechanism for the dissipation of endrin from the environment, since the bulk of what is released is eventually returned to the earth's surface. As was stated previously, pesticides borne by the wind may travel long distances, either as vapors or adsorbed to small particulates, before being redeposited to the hydrosphere and lithosphere as a result of fallout or rainout. Therefore, areas far removed from agricultural lands as well as those nearby are subject to contamination.

The difficulties involved in determining to what extent the contamination of a steam, river, or lake, located in an agricultural area, is due to airborne pesticides are obvious; other forms of contamination (such as surface runoff), operating simultaneously, interfere with analyses. However, one can be reasonably certain that aerial drift following spraying operations, wind-borne transport of pesticide-laden dusts, and rainout of pesticides vaporized from crops and soils have contributed significantly to the contamination of freshwater bodies. While no evidence testifying to the existence of endrin in distant ocean waters has been reported, the presence of related compounds suggests that endrin may be transported similar distances by similar mechanisms (Risebrough et al., 1968; Frost, 1969).

River transport represents one possible mechanism. Pesticides entering rivers from agricultural runoff are subject to codistillation, biodegradation, and sedimentation prior to reaching the sea. Even so, an estimated 1.9×10^3 kg of pesticide reaches San Francisco Bay via the San Joaquin River each year, and 10^4 kg/year is transported to the Gulf of Mexico by the Mississippi River (Risebrough et al., 1968). Surface currents in the ocean, traveling at speeds of up to 100 miles/day, carry these pesticides to areas far removed from their point of origin. The Gulf Stream, for instance, is quite capable of moving pesticides from the coastal areas of the United States to Iceland and the Arctic areas beyond. However, none of these currents is capable of carrying pesticides to Antarctica, and the only route of dispersal able to account for the presence of pesticide residues in the fish and aquatic mammals of Antarctica is atmospheric transport (Frost, 1969). As discussed in Sect. 7.2.2, Risebrough et al. (1968) estimated that approximately 600 kg of dust-borne pesticides was deposited over one particular section of the Atlantic Ocean in a given year. This value clearly underestimated the actual amounts of pesticide delivered to the ocean via the atmosphere, because the method of dust collection used in the study discriminated against materials carried on particles of less than several microns in diameter or present as vapors and subject to rainout. Thus the atmosphere is able to transport significant quantities of pesticides to the open ocean, where river drainage and ocean currents cannot be invoked to explain their presence.

7.4.3 Monitoring for Endrin in the Hydrosphere and Sediment

7.4.3.1 Monitoring for endrin in fresh water and sediment. Pollution surveillance of the surface waters of United States river basins has been in effect since 1957. From 1957 through 1965, monitoring was conducted under the auspices of the Federal Water Pollution Control Administration within the Department of Health, Education, and Welfare. During this period, approximately 6000 samples were collected at more than 100 stations, using a carbon adsorption method (CAM). This method permitted large volumes of water (up to 5000 gal) to be sampled and quantities of contaminant, sufficient for detection by the analytical procedures available at that time, to be recovered. These samples were stored for future reference following analysis.

The advent of highly sensitive analytical technology for chlorinated hydrocarbon insecticides (e.g., electron-capture gas chromotography) rendered possible the analysis of smaller volumes of water. In 1964 and 1965, surveys were performed for selected pesticides at approximately 100 sites, using bottled 1-qt grab samples (Breidenbach et al., 1967). CAM samples were collected at all sites in 1964 but only at sites in the upper and lower Mississippi River basins in 1965. The stations selected for sampling in 1964 and 1965 were predominantly located in areas with known pesticide usage. On the basis of the 1964 survey, old CAM samples were chosen for reanalysis so as to obtain a historic review of pesticide pollution in the surface waters of the United States. The chosen samples also included those representative of areas experiencing pesticide-related fish kills. Analysis of CAM sample extracts and grab-sample extracts was by electron-capture gas chromotography; the lowest detectable concentration was 0.001 μg/liter. Since quantitative adsorption on and desorption from carbon does not always take place with all organic compounds, and since the old CAM method filtered out suspended particulates to which the pesticides may have adsorbed, values from CAM samples must be considered as minimum.

The results of analyses for endrin in CAM samples (1958 to 1965) and in grab samples (1964 and 1965) are presented in Table 7.18, and the ten locations at which the highest levels were observed in grab samples (1964 and 1965) are listed in the first two columns of Table 7.19. The frequency of occurrence in each year by river basin is summarized in Fig. 7.6. Examination of the data for combined CAM samples in Table 7.18 and Fig. 7.6 reveals that fluctuations in both concentration and frequency of occurrence took place during the early years. From 1961 through 1964, the fraction of samples containing endrin progressively increased until, in 1964, 50% of all samples tested were contaminated with this pesticide (Fig. 7.6).

Although some degree of contamination was observed at every river basin studied in at least one year, consistent contamination of a large percent of the samples occurred only in the lower Mississippi basin. In addition, the highest concentrations recorded for CAM samples during the five consecutive years from 1960 to 1964 were all from this area (Table 7.18). A comparative of the frequency of occurrence in grab samples taken in 1964 and 1965 (Fig. 7.6) indicates that the incidence of endrin contamination decreased in all but one river basin in 1965. The exception was the Southeast basin, which remained the same (14% contamination in each year). However, endrin was still present in 60% of the samples from the lower Mississippi basin, which, for the sixth consecutive year, had the highest concentration measured (Table 7.18). In a separate study conducted from July 1964 to July 1965, endrin was detected on three occasions at a concentration of 10 ppb (10,000 ng/kg) in mud from the lower Mississippi River (Novak and Rao, 1965).

During the fall and winter months of 1963, recurrent fish kills took place in the lower Mississippi, and endrin was identified as the causative agent (Anonymous, 1964; Breidenbach et al., 1967; Rowe et al., 1971). The history of endrin contamination in the Mississippi River main stem from St. Paul, Minnesota, to New Orleans, Louisiana, is presented in Fig. 7.7. The occurrence of high concentrations at and below West Memphis from October 1962 through July 1964 is striking. However, a general decrease in concentration took place beginning in the summer of 1964, and no major fish kills were reported on the lower Mississippi River between 1964 and 1966 (Breidenbach et al., 1967).

From 1966 through 1968, monitoring of surface waters for pesticide contamination was conducted by the Federal Water Quality Administration within the U.S. Department of the Interior. Approximately 100 stations were surveyed each year by electron-capture gas chromotographic analysis of grab-sample extracts (Lichtenberg et al., 1970). Endrin was found in approximately 23 and 2% of all samples tested in 1966 and 1967 respectively. No endrin was detected in any sample in 1968. The percent

Table 7.18. Occurrence of positive and presumptive endrin determinations in carbon adsorption method (1958-1965) and in grab samples (1964 and 1965)

Water year (Oct. 1-Sept. 30)	Number samples	Number positive	Maximum concentration (μg/liter)	Water year (Oct. 1-Sept. 30)	Number samples	Number positive	Maximum concentration (μg/liter)
Northeast basin				Southeast basin			
CAM samples				CAM samples			
1958	6	1	0.001	1958	2	1	<0.001
1959	5	0		1959	1	0	
1960	4	0		1960	6	1	0.001
1961	6	1	P	1961	3	0	
1962	7	1	0.001	1962	3	1	P
1963	9	0		1963	14	0	
1964	7	0		1964	10	1	0.001
Grab samples				Grab samples			
1964	7	2	0.025	1964	7	1	P
1965	7	0		1965	7	1	0.015
North Atlantic basin				Ohio, Tennessee, and Lake Erie basins			
CAM samples				CAM samples			
1958	2	1	P	1958	4	0	
1959	1	0		1959	4	0	
1960	7	1	P	1960	3	0	
1961	5	0		1961	6	0	
1962	10	1	P	1962	5	2	P

Table 7.18 (continued)

Water year (Oct. 1–Sept. 30)	Number samples	Number positive	Maximum concentration (µg/liter)	Water year (Oct. 1–Sept. 30)	Number samples	Number positive	Maximum concentration (µg/liter)
1963	9	1	P	1963	11	4	0.012
1964	5	2	P	1964	18	7	P
Grab samples				Grab samples			
1964	8	3	>0.094	1964	12	4	0.015
1965	8	1	0.018	1965	12	4	0.014
Upper Mississippi River basin				Missouri River basin			
CAM samples				CAM samples			
1958	4	0		1958	4	1	P
1959	2	1	0.013	1959	6	0	
1960	4	1	P	1960	6	2	0.004
1961	7	1	0.001	1961	7	2	P
1962	2	0		1962	8	4	0.003
1963	15	11	0.007	1963	8	1	P
1964	12	9	0.004	1964	14	4	0.001
1965	8	0					
Grab samples				Grab samples			
1964	8	5	0.023	1964	11	6	0.026
1965	8	1	0.009	1965	11	3	P

Table 7.18 (continued)

Water year (Oct. 1–Sept. 30)	Number samples	Number positive	Maximum concentration (µg/liter)
Western Great Lakes basin			
CAM samples			
1958	1	0	
1959	3	0	
1960	3	0	
1961	4	0	
1962	3	0	
1963	2	0	
1964	7	2	<0.001
Grab samples			
1964	4	1	0.006
1965	4	0	
Lower Mississippi River basin			
CAM samples			
1958	3	3	0.004
1959	4	3	0.002
1960	3	3	0.064
1961	6	2	0.029
1962	3	3	0.160
1963	12	12	0.214
1964	25	74	0.150
1965	34	22	0.015
Southwest basin			
CAM samples			
1958	1	0	
1959	0		
1960	0		
1961	0		
1962	1	0	
1963	6	6	0.007
1964	10	6	0.002
Grab samples			
1964	6	5	0.014
1965	6	0	
Western Gulf basin			
CAM samples			
1958	4	1	0.008
1959	3	1	0.001
1960	5	1	0.002
1961	4	3	0.008
1962	4	4	0.009
1963	10	3	0.011
1964	7	5	0.004

Table 7.18 (continued)

Water year (Oct. 1–Sept. 30)	Number samples	Number positive	Maximum concentration (μg/liter)
Grab samples			
1964	6	6	0.025
1965	5	3	0.110
Colorado River basin			
CAM samples			
1958	0		
1959	0		
1960	1	0	
1961	0		
1962	1	0	
1963	0		
1964	2	0	
Grab samples			
1964	7	3	0.012
1965	7	1	P
California, Hawaii, and Great Basin			
CAM samples			
1958	0		
1959	0		
Grab samples			
1964	5	2	0.007
1965	6	1	0.014
Pacific Northwest basin			
CAM samples			
1958	3	0	
1959	4	0	
1960	1		
1961	7	1	P
1962	5	0	
1963	12	0	
1964	13	1	P
Grab samples			
1964	10	4	0.019
1965	11	0	
Combined			
CAM samples			
1958	34	8	0.008
1959	33	5	0.013

Table 7.18 (continued)

Water year (Oct. 1-Sept. 30)	Number samples	Number positive	Maximum concentration (µg/liter)	Water year (Oct. 1-Sept. 30)	Number samples	Number positive	Maximum concentration (µg/liter)
1960	0			1960	43	9	0.064
1961	1	0		1961	56	10	0.029
1962	5	0		1962	57	16	0.160
1963	9	1	0.001	1963	117	39	0.214
1964	8	1	P	1964	118	108	0.150
Grab samples				Grab samples			
1964	5	2	0.009	1964	96	44	>0.094
1965	7	1	0.005	1965	99	16	0.116

P = presumptive. Data are reported as presumptive in instances where the results of chromatography were highly indicative but did not meet all requirements for positive identification and quantification.

Source: modified from Breidenbach et al., 1967.

Table 7.19. Top ten locations at which highest levels of endrin were observed

1964		1965		1966		1967	
Location	μg/liter	Location	μg/liter	Location	μg/liter	Location	μg/liter
Potomac River, Great Falls, Md.	0.094	Mississippi River, West Memphis, Ark.	0.116	Hudson River, Narrows, N.Y.	0.069	Kansas River, Lawrence Kan.	0.133
Rio Grande River, El Paso, Tex.	0.067	Atchafalaya River, Morgan City, La.	0.019	South Platte River, Julesburg, Colo.	0.063	Maumee River, Toledo, Ohio	0.086
Big Horn River, Hardin, Mont.	0.026	Delaware River, Trenton, N.J.	0.018	Savannah River, Port Wentworth, Ga.	0.031		
Mississippi River, Vicksburg, Miss.	0.025	Tombigbee River, Columbus, Miss.	0.015	St. Joseph River, Denton Harbor, Mich.	0.029		
Connecticut River, Northfield, Mass.	0.025	Clinch River, Kingston, Tenn.	0.015	Lake Superior, Duluth, Minn.	0.022		
Red River (North), Grand Forks, N.D.	0.023	Rio Grande River, Alamosa, Colo.	0.014	Savannah River, N. Augusta, S.C.	0.022		
Mississippi River, New Roads, La.	0.023	Monongahela River, Pittsburgh, Pa.	0.014	Bear River, Preston, Idaho	0.019		
Yellowstone River, Sidney, Mont.	0.021	Tennessee River, Lenoir City, Tenn.	0.009	Clearwater River, Lewiston, Idaho	0.015		
Columbia River, Clatskanie, Oreg.	0.019	Red River (North), Grand Forks, N.D.	0.009	Connecticut River, Northfield, Mass.	0.014		
Atchafalaya River, Morgan City, La.	0.018	Mississippi River, Delta, La.	0.008	Mississippi River, Delta, La.	0.014		

Source: modified from Lichtenberg et al., 1970.

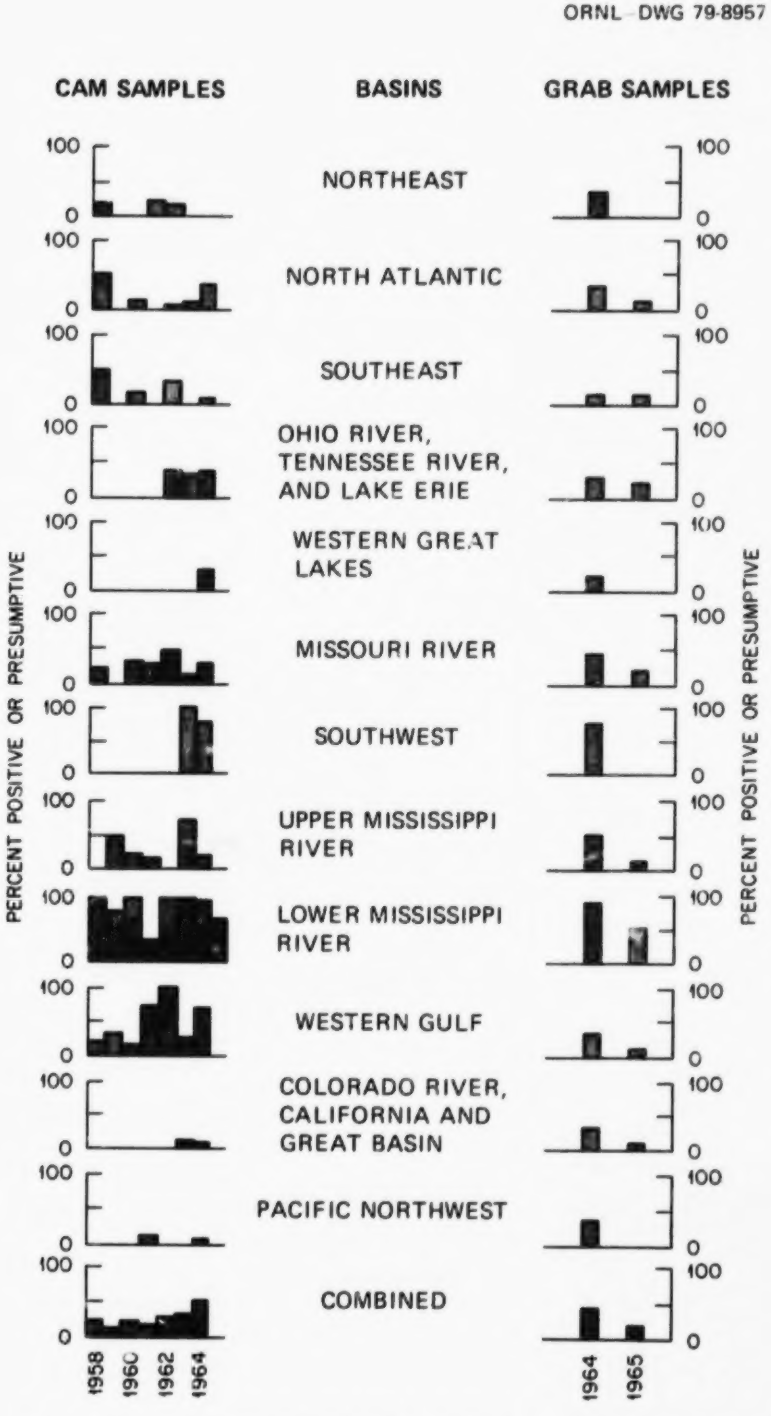

Fig. 7.6. Percent occurrence of endrin in CAM samples, 1958–65, and in grab samples, 1964–65. Source: modified from Breidenbach et al., 1967; Lichtenberg et al., 1970.

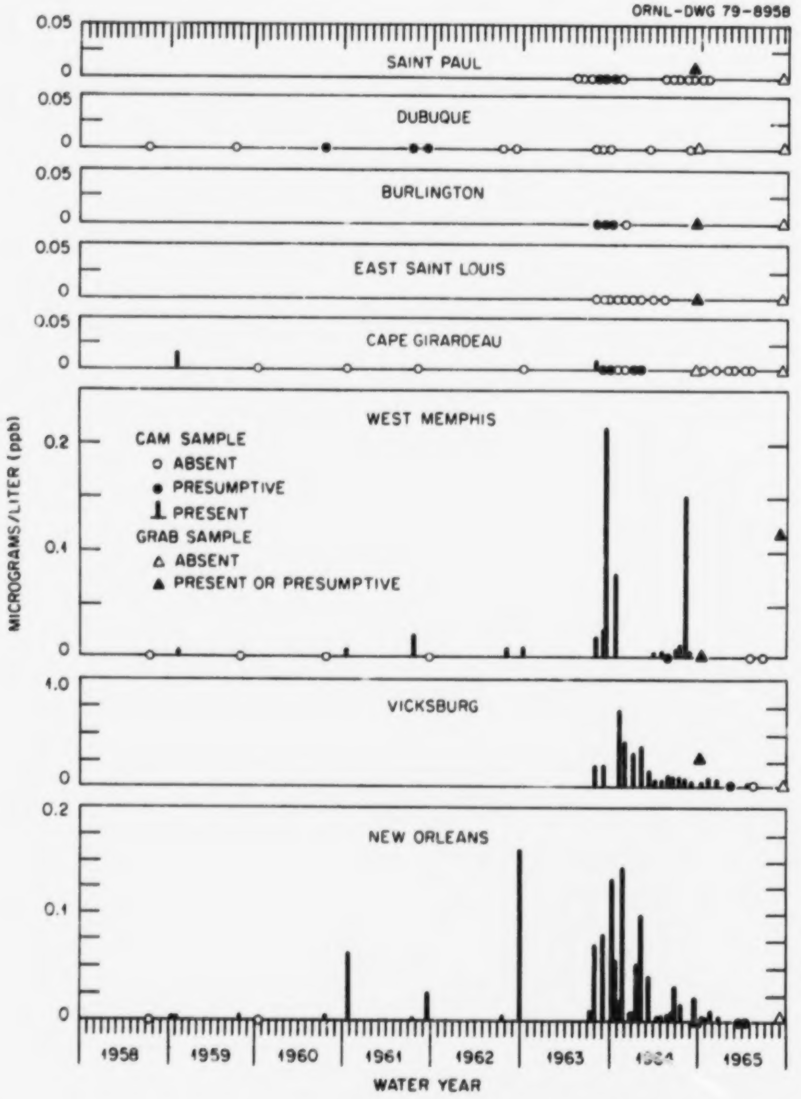

Fig. 7.7. Historical occurrence of endrin in the Mississippi River main stem, CAM samples from water years 1958–65. Source: modified from Breidenbach et al., 1967.

occurrence of endrin in grab samples throughout the United States from 1964 through 1968 is summarized in Fig. 7.8. The locations at which the highest levels were observed in 1966 and 1967 are presented (along with similar data for 1964 and 1965 grab samples) in Table 7.19. The lower Mississippi River still exhibited significant concentrations of endrin in 1966, although it now ranked tenth in contamination as compared with first in 1965. Neither of the two sampling sites at which endrin was observed in 1967 was located in the Mississippi River.

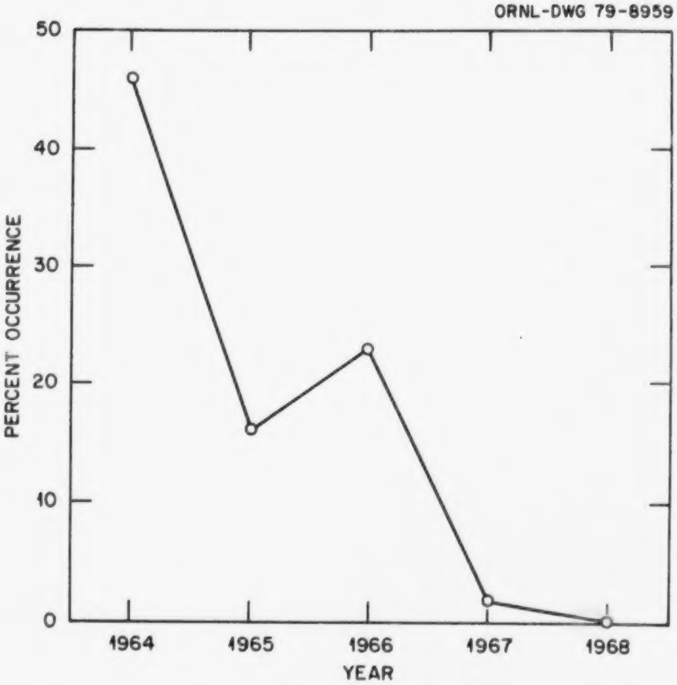

Fig. 7.8. Occurrence of endrin in river basins throughout the United States (grab samples, 1964-68). Source: data as given by Breidenbach et al., 1967; Lichtenberg et al., 1970.

Monitoring of the surface water and sediment of major drainage basins was conducted from 1970 through 1975 by the U.S. Geological Survey within the Department of the Interior, and the results of these surveys were analyzed by the National Water Monitoring Program of the U.S. Environmental Protection Agency (U.S. EPA, 1975b).

The data for 1970 through 1975 (or the last year monitored) were pooled for each drainage basin and are presented in Table 7.20. Unfortunately, the results, as tabulated, cannot be directly compared with those available for previous years, since the number of samples positive for endrin in a given year and the year in which the maximum concentration was observed are not reported. However, some conclusions may be drawn from the data.

Endrin was observed in 10 out of 17 drainage basins at some time between 1970 and 1975. The failure to detect endrin at any site in 1968 was, therefore, only a temporary phenomenon and not indicative of a permanent trend. While the levels of contamination in the lower Mississippi were significant (20 ng/liter), they did not approach the maximum of 214 ng/liter present in 1963 at the time of the fish kills.

Fish kills have, however, been known to occur at similarly low concentrations. In 1961, fish kills attributed to endrin took place

Table 7.20. Endrin residues in whole-water samples (µg/liter) and bottom deposits (µg/kg) in drainage basins of the United States and Puerto Rico

Drainage basins	Whole-water samples: Time period	No. of observations	Arithmetic mean	Standard deviation	Minimum	Maximum	Bottom deposits: Time period	No. of observations	Arithmetic mean	Standard deviation	Minimum	Maximum
1. North Atlantic slope	6/22/70–3/20/75	379	0.0004	0.0053	0.0000	0.1000	6/22/70–1/6/75	226	0.0442	0.2334	0.0000	2.4000
2. South Atlantic slope and eastern Gulf of Mexico	1/12/70–4/3/75	705	0.0013	0.0058	0.0000	0.1000	3/1/70–	571	0.0280	0.1575	0.0000	2.1000
3. Ohio River	1/15/70–4/2/75	235	0.0000	0.0000	0.0000	0.0000	9/21/70–11/14/74	20	0.1000	0.1026	0.0000	0.2000
4. St. Lawrence River	4/17/70–3/26/75	263	0.0002	0.0015	0.0000	0.0200	9/9/70–2/4/75	97	0.0144	0.0520	0.0000	0.2000
5. Hudson Bay and upper Mississippi River	3/5/70–3/19/75	133	0.0000	0.0000	0.0000	0.0000	10/29/70–3/19/75	53	0.0189	0.05745	0.0000	0.2000
6. Missouri River	1/6/70–3/13/75	596	0.0001	0.0004	0.0000	0.0100	8/24/70–3/11/75	58	0.1069	0.1006	0.0000	0.2000
7. Lower Mississippi	1/6/70–3/13/74	920	0.0003	0.0018	0.0000	0.0200	4/28/70–3/11/75	260	0.1285	0.5005	0.0000	6.7000
8. Western gulf of Mexico	1/2/70–11/15/74	1723	0.0001	0.0005	0.0000	0.0200	7/20/70–10/9/74	656	0.0889	0.1217	0.0000	1.9000
9. Colorado River	1/6/70–3/11/75	251	0.0001	0.0006	0.0000	0.0100	10/8/70–8/28/74	20	0.1000	0.1026	0.0000	0.2000
10. The Great Basin	1/2/70–8/15/74	117	0.0015	0.0058	0.0000	0.0400	9/2/70–4/18/74	20	0.1100	0.1021	0.0000	0.2000
11. Pacific slope basins in California	1/29/70–5/31/74	289	0.0001	0.0006	0.0000	0.0100	6/14/71–1/9/74	53	0.1245	0.0979	0.0000	0.2000
12. Pacific slope basins in Washington	2/16/70–5/24/74	63	0.0000	0.0000	0.0000	0.0000	9/2/70–5/23/74	20	0.1000	0.1026	0.0000	0.2000

Table 7.20 (continued)

Drainage basins	Whole-water samples						Bottom deposits					
	Time period	No. of observations	Arithmetic mean	Standard deviation	Minimum	Maximum	Time period	No. of observations	Arithmetic mean	Standard deviation	Minimum	Maximum
13. Snake River	1/13/70–5/6/74	33	0.0000	0.0000	0.0000	0.0000	9/1/70–5/6/74	8	0.1500	0.0926	0.0000	0.2000
14. Pacific slope basins in Oregon and lower Columbia River	1/14/70–4/17/74	31	0.0000	0.0000	0.0000	0.0000	3/22/72–3/22/72	1	0.2000	0.0000	0.2000	0.2000
15. Alaska	9/29/71–9/29/71	1	0.0000	0.0000	0.0000	0.0000	9/29/71–9/29/71	3	0.0667	0.1155	0.0000	0.2000
16. Hawaii	10/13/70–9/28/73	4	0.0000	0.0000	0.0000	0.0000	10/13/70–9/28/73	4	0.1500	0.1000	0.0000	0.2000
17. Puerto Rico	1/13/70–3/20/74	216	0.0175	0.0066	0.0000	0.0200	2/28/73–4/4/74	8	0.0000	0.0000	0.0000	0.0000

Source: U.S. EPA, 1975b.

in three Louisiana streams: a borrow pit canal near Centerville, Bayou L'Onion near Chegley, and the Franklin Canal in Franklin. During these episodes the streams contained 29, 9, and 40 ppt (ng/liter) endrin respectively (Lauer et al., 1966).

The levels present in fish from the lower Mississippi during the summer of 1975 were apparently high enough to result in the death of 80% of Louisiana's brown pelican population. All of the birds examined contained lethal doses of endrin in their tissues (Anonymous, 1975c; King and Flickinger, 1977).

Although endrin contamination of the surface waters of the lower Mississippi has decreased since 1964, the large-scale use of endrin in the agricultural lands surrounding this basin prior to that period is reflected in the levels of the pesticide detected in bottom deposits. The highest concentration of endrin in the sediment of any river basin (6700 ng/kg) between 1970 and 1975 was found in the lower Mississippi. This value has, however, decreased since 1964-65, when a concentration of 10,000 ng/kg was reported.

The basins experiencing the highest levels of contamination in surface water from 1970 to 1975 (100 ng/liter) were the North Atlantic slope and the South Atlantic slope-eastern Gulf of Mexico. The North Atlantic basin had demonstrated similar levels of contamination (>94 ng/liter) in 1964 (Table 7.18). Levels of endrin in the bottom deposits of the North Atlantic slope and South Atlantic slope-eastern Gulf of Mexico basins were 2400 and 2100 ng/kg respectively. Except for the western Gulf of Mexico (1900 ng/kg) and the lower Mississippi basin (previously discussed), these levels were ten times greater than those observed in the bottom deposits of all other basins.

Monitoring for pesticides in the surface water and sediment of Louisiana waterways has recently been conducted by the U.S. Army, New Orleans District Corps of Engineers, as part of a program to assess the impact of proposed maintenance dredging on the water quality in the area of the work (U.S. Army, 1975). Endrin was not detected in any of the surface water samples but was found at concentrations ranging from 0.00 to 2.00 µg/kg in the bottom sediments of various waterways. The exact locations at which endrin was found and the concentration range for each location are presented in Table 7.21.

From fall 1965 through fall 1971 the U.S. Geological Survey monitored selected streams west of the Mississippi for pesticide contamination. In the 1965-66 survey, 11 streams, located within agricultural areas, were chosen for investigation. From 1967 through 1971, the number of streams monitored was increased to 20, including the original 11. Grab samples consisting of water and suspended sediment were analyzed by electron-capture gas chromatography with a limit of detection of 5 ppt. The streams which were positive for endrin are presented in Table 7.22. In 1965-66, endrin was detected once at each of four sites and three times at one site. The location of the last site was the Rio Grande River below Anzulduos Dam, Texas. Concentrations

Table 7.21. Endrin in bottom sediments of Louisiana waterways-1975

Location	Concentration range (μg/kg)
Atchafalaya Bay	0.00–0.40
Atchafalaya basin, east access channel	0.00–<0.10
Lower Atchafalaya River, Berwick Lock entrance canal	0.00–2.00
Upper Atchafalaya River, Old River Lock tailbay	0.00–0.10
Gulf Intercoastal Waterway, Morgan City to Port Ellen	0.00–0.5
Gulf Intercoastal Waterway, Atchafalaya River to Sabine River	0.00–0.20
Mississippi River, Baton Rouge to the Gulf	0.10–1.40
Calcosieu River	0.00–0.20
Bayou Le Carpe	0.00–0.10

Source: U.S. Army, 1975.

Table 7.22. Endrin concentrations in selected western streams 1965-71

Station	Date	Concentration (ppt or ng/liter)
Colorado River at Wharton, Texas	10/22/65	5
	11/30/67	70
Colorado River at Yuma, Arizona	6/02/66	15
	6/01/67	10
Rio Grande, below Anzalduos Dam, Texas	2/16/66	10
	5/17/66	40
	6/17/66	25
Missouri River at Nebraska	5/17/66	35
Snake River at King Hill, Idaho	2/17/66	25
Brazos River at Richmond, Texas	7/30/68	20
Gila River at Gillespie Dam, Arizona	11/21/67	10
	9/17/69	30
	10/13/69	10
	2/17/70	10

Source: modified from Brown and Nishioka, 1967; Manigold and Schulze, 1969; Schulze et al., 1973.

at all sites ranged from 5 to 35 ppt (Brown and Nishioko, 1967). From 1966 through 1968, endrin was detected once at each of four sites with concentrations ranging from 10 to 70 ppt (Manigold and Schulze, 1969). From 1968 through 1971, endrin was detected three times at one location, the Gila River at Gillespie Dam, Arizona. Concentration ranged from 10 to 30 ppt (Schulze et al., 1973).

Other freshwater bodies monitored for endrin in the United States include Lake Mead, Arizona (Lyons and Salman, 1972); Lake Poinsett, South Dakota (Hannon et al., 1970); and Everglades National Park, Florida (Kolipinski et al., 1971). Endrin was not detected at any of these sites. Samples of bottom sediment from three locations in the Bid Muddy Creek watershed, Kentucky, showed endrin to be present in quantities less than 0.01 ppm (Golden and Twilley, 1976).

Municipal water supplies fed by contaminated streams are themselves subject to contamination, and endrin has been detected in the raw and finished water of several treatment plants. All studies were conducted in areas with a history of high endrin usage and where mass fish kills, due to endrin, had occurred. One such treatment plant was located on Bayou Teche in the city of Franklin, Louisiana (Lauer et al., 1966). Synchronous carbon adsorption samples were collected from the raw-water pump on Bayou Teche, from a pump located between the open-air holding reservoir and the treatment plant, and from a finished waterline at the treatment plant, continuously from May 1961 through January 1962 and intermittedly from that time through May 1962. Water treatment consisted in prechlorination, alum-lime coagulation, use of activated charcoal at the rate of 1 to 2 ppm, sedimentation, rapid sand filtration, chlorine disinfection, and softening. The results are presented in Table 7.23.

Table 7.23. Concentration of endrin recovered by the carbon adsorption method from the Franklin Municipal Treatment Plant on Bayou Tech, Louisiana, 1961-62

	Number of samples analyzed	Number of samples containing endrin	Concentration (ppt)	
			Range of positive samples	Median of positive samples
Raw water (pumphouse)	22	7	1-35	7
Raw water (holding reservoir)	21	5	1-12	3
Finished water (treatment plant)	18	2	2-23	11

Source: G. J. Lauer, H. P. Nicolson, W. S. Cox, and J. I. Teasley, *Trans. Am. Fish. Soc.* 95(3): 310-316 (1966). Copyright 1966 American Fisheries Society.

Despite the rigorous treatments, endrin was recovered from the finished water going to the consumer as well as from the raw river water and the raw water after storage. In fact, the concentration in one contaminated sample of finished water (23 ppt) exceeded that of the most contaminated sample of stored raw water (12 ppt). Whether this phenomenon was due to an error in sampling or analysis or whether further contamination occurred during the treatment process itself remains unknown. All samples containing detectable quantities of endrin were collected from early June through late November 1961. The highest value (35 ppt) occurred in raw water from a late July sample. Dead fish floated past the Franklin water intake for several days during that period.

In a study conducted between March 1964 and June 1967, over 500 grab samples of finished drinking water and corresponding raw water were collected from ten selected municipal water treatment plants whose source was either the Mississippi or the Missouri River (Schafer et al., 1969b). Grab samples consisting of 100 ml in 1964, 100 ml and 1 gal (3.785 liters) in 1965, and 3.785 liters in 1966-67 were collected and analyzed by electron-capture gas chromotography. The decision to use a larger sampling volume resulted from the poor precision obtained with the 100-ml aliquots. The results are presented in Table 7.24.

Of the 458 finished water samples assayed between 1964 and 1967 by both methods, 156 (34%) contained detectable concentrations of endrin. However, the number of finished water samples containing concentrations of endrin in excess of 0.1 ppb, which is a suggested maximum reasonable stream allowance (Schafer et al., 1969b), decreased from 23 (10%) in the period 1964-65 to zero in the period 1966-67.

The most recent study of endrin contamination of drinking water was conducted by the U.S. Environmental Protection Agency (1974a). Endrin was detected in the finished water from the Carrollton Water Plant in New Orleans, Louisiana. The highest concentration measured was 4 ppt. The results of similar investigations at the Jefferson Parish No. 1 water plant (Metairie, Louisiana) and the Jefferson Parish No. 2 water plant (Marerro, Louisiana) were not reported.

Approximately five months following a single application of endrin to Louisiana sugarcane fields on July 6, 1967, endrin was detected in the groundwater (Willis and Hamilton, 1973). Concentrations decreased from 0.17 ppb to 0.04 ppb over the ensuing three-month period. Endrin was also detected in the groundwater following a similar application in July 1968. In this case the endrin appeared at a concentration of 0.78 ppb immediately following treatment and decreased to a concentration of 0.25 ppb after one month. In both years the occurrence of endrin in groundwater could be correlated with rainfall and runoff following periods of drought which had caused numerous deep cracks in the soil. It is likely that the pesticide, adsorbed to either carrier granules or soil particles, was washed downward into these cracks and was subsequently transported to the groundwater supply. When rainfall occurred 72 hr following application, contamination of the groundwater amounted to 0.07 ppb; if precipitation followed 24 hr after application, the groundwater contained 0.45 ppb. Thus, heavy rains occurring immediately after endrin application and following

Table 7.24. Endrin in drinking water and associated river water

1-gal samples of water				100ml samples of water			
Location	Date	Ratio[a]	Number of samples with concentrations >0.1 ppb	Location	Date	Ratio[a]	Number of samples with concentrations >0.1 ppb
				Finished drinking water			
Algiers	03/31/65 08/25/65	4/12	0 0	Algiers	03/03/64 12/17/74	14/19	1
Carrolton	03/31/65 08/25/65	3/12	0	Carrolton	03/03/64 12/17/64	14/20	2
Carville	03/29/65 12/14/67	1/19	0	Carville	04/16/64 06/08/66	24/39	3
Vicksburg	03/29/65 05/25/67	7/45	0	Vicksburg	03/31/64 06/30/66	34/41	6
Cape Girardeau	04/19/65 06/07/67	3/39	0	Cape Girardeau	06/02/64 03/14/66	11/25	3
Omaha	05/04/65 05/23/67	1/19	0	Burlington	06/30/64 03/22/66	4/10	1
St. Louis	12/11/66 05/25/67	1/7	0	St. Louis	06/11/64 06/16/66	10/32	1
Burlington	03/24/65 08/06/67	0/11	0	Jefferson City	06/04/64 07/16/64	6/7	0
Jefferson City	05/30/66 05/26/67	0/15	0	Kansas City	06/11/64 05/23/66	5/12	3
Kansas City	03/19/65 05/23/67	4/54	0	Omaha	06/01/64 03/07/66	10/20	3
Total		24/233	0	Total		132/225	23
				Raw water			
Carville	12/14/66 01/05/67	0/2	0	Carville	06/17/64 03/10/66	3/10	1
Vicksburg	02/27/67 05/25/67	1/4	0	Cape Girardeau	06/02/64 03/14/66	4/12	3
Cape Girardeau	03/29/65 06/07/67	2/8	0	Kansas City	04/27/66 05/23/66	0/2	
Jefferson City	05/30/66 05/26/67	0/15	0				
Kansas City	05/04/66 05/23/67	1/22	0				
Omaha	04/20/66 05/23/67	1/16	0				
Total		5/67	0	Total		7/24	4

[a] Ratio of number of samples with detectable concentrations <0.1 ppb to total number of samples.

Source: modified from Schafer et al., 1969b.

a prolonged drought will favor contamination of the groundwater supply and potentially the drinking water supply.

Endrin contamination of freshwater bodies in Ontario, Canada, has also been reported (Miles and Harris, 1973). In 1971, in order to compare residue contributions from areas with differing degrees of pesticide usage, a study was conducted in which three streams draining an agricultural, an urban-agricultural, and a resort area, respectively, were monitored. Both water (including suspended particulates) and bottom sediment were sampled from mid-April to mid-October 1971 and analyzed by electron-capture gas chromotography. The results are presented in Table 7.25.

Table 7.25. Endrin concentration in water and bottom mud from three streams in Ontario, Canada — 1971

Date	Concentration in water (ppt)	Date	Concentration in bottom mud (ppb)[a]
	Big Creek (agricultural)		
Apr. 13	<1	Apr.	<0.2
26	<1	May	0.2
May 4	<1	June	<0.2
11	<1	July	<0.2
25	1	Aug.	<0.2
June 8	3	Sept.	0.2
22	7	Oct.	0.3
July 26 to Oct. 12[b]			
Average	1		
Range	<1-3		
	Thames River (urban-agricultural)		
Apr. 15	-	May	-
26	-	June	-
May 5	-	July	-
12	-	Aug.	-
26	-	Sept.	-
June 9 to Sept. 15[b]		Oct.	-
Average	-		
Range	-		
Sept. 29	-		
Oct. 13	-		
28	-		
	Muskoka River (resort)		
May 5	2	May	0.3
26	<1	June	<0.3
June 10	<1	July	<0.3
June 23 to Nov. 4[b]		Aug.	<0.3
Average	<1	Sept.	<0.3
Range	<1-1		

[a] Because concentrations were very uniform, results for each month have been averaged for presentation in this table.

[b] Because of the leveling off of insecticide residues at lower concentrations for these uniform periods, data have been condensed by reporting average concentrations and ranges.

- = not detected.

= qualitative identification of the compound was made but the amount was less than the lowest level of reporting.

Source: modified from Miles and Harris, 1973.

Endrin was present in both the water and bottom mud of Big Creek, which drains 280 square miles of primarily tobacco farms. Concentrations in water ranged from <1 to 7 ppt, while concentrations in sediment ranged from <0.2 to 0.3 ppb. An earlier survey of this region reported endrin absent in both water and sediment; but in this case the limits of detection were 3 ppb (Miles and Harris, 1973).

The Thames River, which drains the urban-agricultural city of London, showed no evidence of endrin contamination in either water or bottom mud (Miles and Harris, 1973). However, endrin was detected at concentrations of less than 0.01 ppm in fish from this river and must therefore have been present at low concentrations in the water and/or sediment.

Surprisingly, the resort area of Muskoka Lakes exhibited endrin concentrations equal to or in excess of those detected in the agricultural area. Concentrations in water ranged from less than 1 to 2 ppt; concentrations in sediment ranged from less than 0.3 to 0.3 ppb. No explanation was proposed for these unexpected results (Miles and Harris, 1973).

Another study (Stewart et al., 1977) of two New Brunswick streams draining agricultural land and analyzed for organochlorine and organophosphorus insecticide residues indicated as much as 0.45 ppm endrin in bottom sediments.

Although concentrations of endrin in fish are not directly proportional to those in water, the presence of endrin in fish is indicative of hydrospheric contamination. Additional information concerning the locations of problem areas may be obtained by consulting Sect. 5.2.2.

7.4.3.2 Monitoring for endrin in estuarine water and sediment. Estuaries receiving water from rivers draining agricultural lands are susceptible to contamination by insecticides. Due to the westerly tide, which may carry Mississippi River water into Barataria Bay, the possibility of contamination of the surrounding estuarine area by endrin was considered likely. A study was conducted from October 1968 through May 1969 in which water, sediment, and oysters from Grand Bayou, Hackberry Bay, and Creole Bay (all adjacent to Barataria Bay) were sampled and analyzed for the presence of endrin by electron-capture gas chromatography (Rowe et al., 1971). This study area, located some 40 miles south of New Orleans, eventually drains the entire agricultural watershed to the north.

Water samples from all stations on every sampling date contained less than 1 ppb endrin, and the maximum concentration of endrin detected in bottom sediment was less than 5 ppb. Sixty-nine percent of the oyster samples analyzed were positive for endrin. The highest concentration observed was less than 6 ppb. Comparison of endrin levels in oysters with those from a similar study conducted in 1965-66 reveals that influx of endrin into the study area decreased between 1966 and 1968 (Rowe et al., 1971). The 1968-69 maximum endrin concentration was 29 times lower than that observed in 1965-66. These results are reasonable in light of the failure to detect any endrin in the lower Mississippi basin during the 1968 survey (see Sect. 7.4.3.2).

Monitoring of estuarine waters for endrin by analysis of shellfish was conducted along the coast of California in 1967 (Modin, 1969). Nine estuaries from Humboldt Bay to Hedionda Lagoon were sampled. Endrin was found in the Asiatic clam (*Corbicula fluminea*) at only one site, West Island in San Francisco Bay. The highest concentration detected was 10 ppb.

A study was conducted from September 1969 to June 1970 to determine the levels of endrin in Arkansas Bay, Texas, and its adjacent contributing bays (Fay and Newland, 1972a). The land surrounding these bays is primarily ranchland with secondary agricultural usage. Samples of water, sediment, and eight types of organisms were analyzed by electron-capture gas chromotography. Endrin was detected in only 1 out of 80 water samples from Arkansas Bay at a concentration of 4.4 ppb. No endrin was found in either sediment or organisms.

Monitoring of estuarine waters by analysis of mollusks was conducted in 15 coastal states from 1965 through 1972 (Butler, 1973). Endrin was found at concentrations above 5 ppb only in organisms from California and Texas. Endrin appeared in 14 out of 772 samples collected from California waters. The maximum concentration observed was 19 ppb and was found in *Crossostrea gigas* from Elkhorn Slough in 1968. In Texas, out of 728 samples, endrin appeared in 22. The highest value was 32 ppb and was found in *Crossostrea virginica* from the Arroyo Colorado in 1965.

7.4.4 Mechanisms for Removal of Endrin from the Hydrosphere and Sediment

In June 1959 a fish kill occurred in a pond located in Weld County, Colorado. Dead and dying fish were observed several days following the aerial application of endrin to a nearby beetfield. Samples of water, mud, and vegetation were taken from the pond and analyzed for the presence of endrin (Bridges, 1961). The concentration of endrin in water four days after spraying was 40 ppb. After 23 days the concentration had decreased to 4 ppt. By the 31st day, endrin could no longer be detected in water samples. The endrin concentrations in vegetation and mud 16 days after spraying were 250 and 170 ppb respectively. Endrin was no longer detected in vegetation 51 days after spraying or in mud 70 days after spraying. Even though the analytical technique employed in this study (paper chromatography) is not as sensitive as others and might not have detected very low levels of residual contamination, rapid reduction in concentration appears to have occurred.

Several mechanisms for the removal of pesticides from the hydrosphere are known (Chesters and Konrad, 1971). Volatilization and codistillation most likely accounted for a large proportion of the losses occurring in the situation described above. The pond in question consisted of four well-exposed acres with an average depth of 3 ft. Significant wind action was observed during the summer sampling period (no wind velocities were given), and water temperatures averaged 24°C (75°F). The combination of large surface area, shallow depth, warm temperatures, winds, and lack of vegetative cover provided the ideal setting for maximum evaporative loss.

Dissipation of pesticides from the environment occurs as a result of biochemical, photochemical, and thermal degradation. However, the degradation products themselves may be of equal or greater toxicity and just as persistent as the parent compounds; under such circumstances, degradation would result in little benefit to the environment.

Endrin is known to undergo rearrangement reactions under the influence of both sunlight and heat when treated either in the solid state or as a solution in an organic solvent such as hexane (Sect. 2.3). All of the rearrangement products studies have proved to be less toxic to test animals than endrin itself (Soto and Deichmann, 1967). From an environmental point of view, such rearrangements are therefore desirable. Similar reactions are believed likely when endrin is adsorbed to particulates (as when present in the hydrosphere) (Klein and Korte, 1967); however, studies in support of this prediction have not been reported. No photolytic reaction was observed when endrin, at a concentration of 10 μg per liter of distilled water, was exposed to both sunlight and artificial fluorescent light for a period of three weeks (Eichelberger and Lichtenstein, 1971).

Degradation of endrin by aquatic microorganisms has been reported and probably contributes at least to some extent to the dissipation of that pesticide from the aquatic environment (Sect. 3.2). Microbial degradation appears to be most important in freshwater systems.

Due to their hydrophobic, lipophilic nature, organochlorine insecticides adsorb onto particulate matter suspended in water and are transported to the sediment. The association of pesticides with suspended particulates is borne out by comparative studies of ponds during periods of murky water and clear water following a settling period. Measurable pesticide residues in the water often drop to nondetectable levels within a week (Iverson, 1967). Evidence that endrin behaves in this predicted fashion was provided by the work of Pfister et al. (1969). Particulate matter from a sample of water collected from the western basin of Lake Erie was layered onto a sucrose gradient and centrifuged. The gradient was subsequently divided into portions and analyzed for the presence of organochlorine insecticides. Endrin was found attached to the less dense upper fractions, which consisted of organics, detritus, and microorganisms. All positive samples appeared to be associated with particles equal to or greater than 0.15 μm in diameter. The highest concentration observed was 9.6 ng per liter of water. No endrin was detected in the raw lake water following the centrifugation procedures used to separate the particulates.

In an environment such as Lake Erie, where the shallow waters are easily disturbed by wind action, the turnover and accumulation of pesticides in bottom sediments may be quite significant.

Adsorption is an indirect form of dissipation from the hydrosphere, since the availability of the pesticides for uptake by plants and aquatic organisms is markedly reduced by the process. However, biochemical degradation and rates of volatilization are also reduced, leading to the conclusion that adsorption and sedimentation result in a safer but more persistent form of contamination.

When natural processes are insufficient, it is possible to remove trace amounts of endrin from water by the use of large quantities of activated carbon (Chesters and Konrad, 1971). An example of such a situation was reported by Stoltenberg (1972). In June 1971 a man deliberately dumped 0.5 gal of a mixture of endrin and strychnine into Shawnee Lake, a three-acre reservoir in the Shawnee State Forest near Portsmouth. Within one day, 4000 fish along with other aquatic organisms in the lake had died. Concentrations of endrin ranged as high as 15 ppb, 25 times greater than the concentration needed to kill minnows in 43 hr. With the aid of a controlled activated-carbon filtration system, 98 to 99% of the endrin was removed from the lake. Removal of pesticides from large bodies of water is not feasible.

7.5 ENDRIN IN THE LITHOSPHERE

7.5.1 Introduction

Since a large proportion of the endrin entering the environment is initially released to the soil, the fate of endrin in the soil determines, to a great extent, the degree to which the rest of the environment will become contaminated. Depending upon conditions, the soil may act as either a barrier or a link in the physical transfer of endrin from one point in the environment to another (Adams, 1967). Some types of soil adsorb endrin and prevent it from escaping to the atmosphere, while others show little affinity for the pesticide and permit extensive volatilization. The situation is even more complicated when the variety of climates and topographies which may enhance or reverse the restraining effects of soil retention is considered. For example, rainfall on sloping lands may result in surface erosion leading to transport of endrin to the hydrosphere despite its being tightly adsorbed by the soil.

Many mechanisms by which endrin can be dispelled from the environment also transpire in the lithosphere. It is in the field that granular endrin may undergo photodecomposition through exposure to natural sunlight (Sect. 2) or where anaerobic conditions favorable for microbial degradation (Sect. 3) may develop.

Whether endrin undergoes transportation from the soil, degradation in the soil, or retention by the soil depends upon so large a number of factors that the formulation of hard-and-fast generalizations becomes exceedingly difficult. The availability of extensive soil monitoring data has aided in identification of seriously contaminated areas and has revealed the levels of residues in the vicinity of sites where endrin-related environmental disasters have occurred. Residue levels in root crops and soil invertebrates may also be an effective indicator of soil contamination (Sect. 8).

7.5.2 Sources of Endrin in the Lithosphere

The major source of endrin in the lithosphere is direct application. Endrin may be applied to the soil per se or may reach the soil indirectly following treatment of crops. The quantities of endrin used and the number of acres treated during 1964, 1966, and 1971 are summarized by crop in Table 7.26 and by region in Table 7.27. The regions are presented pictorially in Fig. 7.9 (Eichers et al., 1968; Eichers et al., 1970; Andrilenas, 1974). Although endrin usage was apparently widespread, especially in 1964, the largest single use of endrin in the United States during each of these years was for the control of lepidopterous larvae attacking cotton crops in the southeastern and delta states. In 1971, 75% of all endrin used was applied to cotton, while 99% of the total usage was confined to the southeastern and delta states. When the quantity of endrin applied to cotton in 1971 (4.849×10^5 kg) is compared with that applied in 1964 (8.467×10^5 kg), it appears as though a 43% reduction in endrin usage had occurred. These figures are, from one standpoint, quite misleading in that the number of acres treated in 1971 (2.62×10^5) was only 22% of the number treated in 1964 (1.194×10^6), so that the number of pounds applied per acre increased by a factor of 2.6. Thus, while contamination of the lithosphere was less diffuse in 1971 than in 1964, pockets of more extensive contamination were created. The marked

Table 7.26. Quantities of endrin used on specified crops and number of acres treated (by crop)

Crop	1964		1966		1971	
	1000 acres	1000 pounds	1000 acres	1000 pounds	1000 acres	1000 pounds
Corn	9[a]	4			75	30
Cotton	1194	1865	403	510	262	1068
Soybeans	48	9			58	23
Tobacco	149	150	24	20		
Other field crops	49	12	55	17	193	256
Irish potatoes	141	61			3	5
Other vegetables	10	20			1	1
Apples	30	30	22	13	9	2
Other fruits and nuts	[b]	[b]	18	175	47	33
Total	1630	2125	522	735	648	1418

[a] Less than 10,000 acres treated; 9000 assumed.

[b] Not reported.

Source: modified from Eichers et al., 1968; Eichers et al., 1970; Andrilenas, 1974.

7.27. Quantities of endrin used on all crops and number of acres treated (by region)

Region	1964		1966		1971	
	1000 acres	1000 pounds	1000 acres	1000 pounds	1000 acres	1000 pounds
Northeast	40	13	12	5	7	5
Lake states	60	12		[b]		
Corn belt	4.5[a]	1.5			127	37
Northern Plains	4.5[a]	1.5			6	4
Appalachian	168	155	47	194		
Southeast	218	484	31	67	156	339
Delta states	628	893	358	435	273	990
Southern Plains	291	231	71	34		
Mountain	80	42	3	<0.5[c]	26	7
Pacific	136	318		53	36	
Total	1630	2151	522	735	648	1418

[a] Less than 10,000 acres treated; source estimates 4500 acres treated with 1500 lb of endrin.

[b] Not reported.

[c] Not included in total.

Source: modified from Eichers et al., 1968; Eichers et al., 1970; Andrilenas, 1974.

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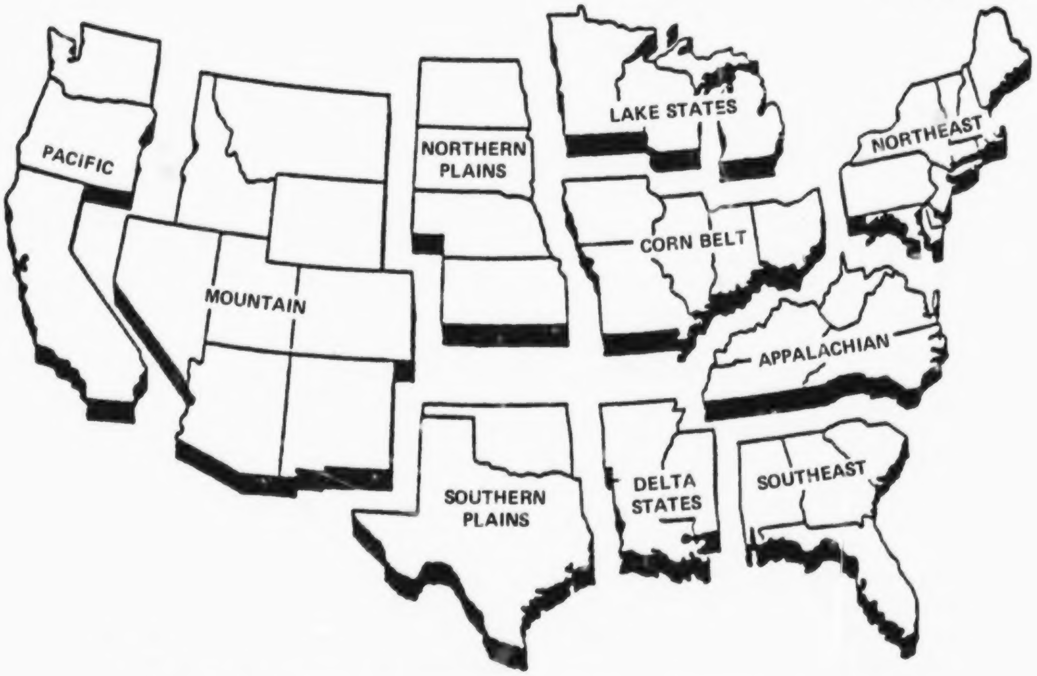
PACIFIC
MOUNTAIN
NORTHERN PLAINS
LAKE STATES
NORTHEAST
CORN BELT
APPALACHIAN
SOUTHERN PLAINS
DELTA STATES
SOUTHEAST

Fig. 7.9. Farm production regions. Source: modified from Andrelinas, 1974.

decrease in the use of endrin during 1966 may have resulted from variations in the weather, pest infestations, and crop production during that year. The massive fish kills attributed to endrin in the early sixties may also have contributed to a temporary decline in use of the pesticide during that year.

The amount of endrin reaching the soil during treatment depends upon the method of application, the type of crop, and the type of soil. Four examples are presented in Table 7.28 (Harris et al., 1966). When endrin was applied to tall dense crops (such as tobacco at site 1), residues did not appear in the soil. Application of endrin directly to the soil (as in sites 2 and 3) only resulted in trace amounts actually contaminating the soil, since both of these soils have limited retentive ability for endrin, and dissipation of the pesticide by wind or runoff readily occurs.

Table 7.28. Endrin in soil as a function of soil type, crop, and mode of application

Site	Soil type	Crop	Mode of application	Year applied	Detected (late 1964) (ppm)
1	Sandy loam	Tobacco, rye	Foliar	1963	None
2	Clay loam	Sugar beets	Surface	1963	<0.1
3	Clay	Sugar beets	Surface	1964	<0.1
4	Muck	Radishes	Surface	1960-1964	3.8

Source: Reprinted with permission from M. L. Schafer et al., *J. Agric. Food Chem.* 14(4): 398-403 (1966). Copyright 1966 American Chemical Society.

The most serious problem arises when endrin is applied directly to a soil with high organic content such as muck (site 4). In this case the pesticide is adsorbed relatively quickly, clings tenaciously, and is only removed with difficulty.

Fallout and rainout of airborne particulate matter to which endrin has adsorbed are important mechanisms for contamination of nonagricultural soil or soil on which endrin, specifically, has never been used. These mechanisms have been discussed in detail in the sections dealing with removal of endrin from the atmosphere (Sect. 7.3.4) and sources of endrin in the hydrosphere (Sect. 7.4.2).

In one study (Abbott et al., 1965), rainwater was examined for traces of pesticides. Endrin was not detected; but the quantities of those pesticides which were detected were insufficient to account for more than a very small percentage of their representation in untreated soils. Although the concentrations of these compounds in air were lower by a factor of 10 to 100 than that in rainwater (Abbott et al., 1965), the vastly greater volume of air which passes over the soil may have rendered its contribution of greater significance. Along with the constantly changing contact between air and soil, the mass of air within the surface soil may alter by up to 5% due to variations in temperature throughout the day. Thus the soil may act as a gas-solid chromatographic "column," and, assuming a tendency toward equilibrium, treated soils will lose pesticides, while untreated soils will gain them.

Wind drift and irrigation with contaminated water are other possible routes of endrin to the soil. They are also discussed in Sect. 7.4.2, which deals with sources of contamination of the hydrosphere. Small amounts of endrin may also be transported to untreated soils by migration of contaminated soil invertebrates, primarily earthworms. The extent to which these organisms bioaccumulate endrin is discussed in Sect. 8.2.

7.5.3 Monitoring for Endrin in the Lithosphere

A comprehensive program for monitoring levels of pesticide residues in soils of the United States (the National Soils Monitoring Program, NSMP) was initiated in 1969 as part of the National Pesticide Monitoring Program. Approximately 25% of some 13,300 officially designated sites are surveyed each year (Carey et al., 1973). The results for the first two years (1969 and 1970) have been reported and comprise a baseline against which future trends may be evaluated (Crockett et al., 1971; Wiersma et al., 1972a). In 1969, both cropland and noncropland soil was sampled from 43 and 11 states respectively. The 1970 study was limited to cropland soil, and only 35 states were included. Soil samples were extracted with hexane-isopropanol and analyzed for organochlorine insecticides by electron-capture gas chromatography. The limit of detection for endrin was 0.01 ppm in dry soil sample. The data for the cropland soil surveys are presented in Tables 7.29 (by state) and 7.30 (by cropping region). No endrin was detected in the noncropland sites investigated in the earlier study.

Of the 1729 sites sampled in 1969, 39 (2.3%) contained endirn residues. The mean residue level was less than 0.01 ppm, with a range of 0.00 to 0.56 ppm.

In 1970, 27 (1.79%) of the 1506 sites sampled were positive for endrin. The mean residue level was once again less than 0.01 ppm, with values ranging from 0.00 to 0.90 ppm. Although endrin was not detected in soils from Indiana, Iowa, or South Dakota in either year, an independent survey of 400 NSMP sites in the corn-belt region, conducted in 1970, detected endrin in one site in each of these states. The concentrations reported were 0.02, 0.02, and 0.04 ppm for Indiana, Iowa, and South Dakota respectively (Carey et al., 1973).

Ketoendrin was detected at nine (0.5%) of the sites sampled in 1969 at a mean concentration of less than 0.01 ppm (range of 0 to 0.13 ppm), while endrin aldehyde was detected in only one sample in the same year at a concentration of 0.03 ppm.

Table 7.29. National Soils Monitoring Program for endrin residues: cropland soil FY 1969 and FY 1970

State	Year	Number of sites analyzed	Number sites positive	Sites positive (%)	Mean residue level (ppm)	Maximum level detected (ppm)
Alabama	1969	22	2	9.1	<0.01	0.05
	1970	21	2	9.52	0.01	0.16
Arizona	1969	8	3	37.5	0.07	0.22
	(endrin ketone)	(8)	(2)	(25)	(0.01)	(0.07)
	1970	NS				
Arkansas	1969	47	5	10.6	0.01	0.29
	(endrin ketone)	(47)	(2)	(4.3)	(<0.01)	(0.13)
	1970	47	3	6.38	<0.01	0.03
California	1969	65	9	13.9	0.01	0.16
	1970	65	5	7.69	<0.01	0.10
Colorado	1969	60	3	5.0	<0.01	0.02
	(endrin ketone)	(60)	1	1.7	<0.01	0.05
	1975	NS				
Connecticut	1969	2	ND			
	1970	20a	ND			
Delaware	1969	3	ND			
	1970	NS				
Florida	1969	18	2	11.1	0.03	0.38
	(endrin aldehyde)	(18)	(1)	(5.6)	(<0.01)	(0.03)
	(endrin ketone)	(18)	(1)	(5.6)	(<0.01)	(0.03)
	1970	17	1	5.88	0.05	0.90

Table 7.29 (continued)

State	Year	Number of sites analyzed	Number sites positive	Sites positive (%)	Mean residue level (ppm)	Maximum level detected (ppm)
Georgia	1969	22	1	4.6	0.02	0.42
	1970	28	ND			
Idaho	1969	33	ND			
	1970	NS				
Illinois	1969	142	ND			
	1970	140	ND			
Indiana	1969	78	ND			
	1970	78	ND			
Iowa	1969	151	ND			
	1970	150	ND			
Kentucky	1969	31	ND			
	1970	30	ND			
Louisiana	1969	27	1	3.7	<0.01	0.06
	(endrin ketone)	27	1	3.7	<0.01	0.02
	1970	26	2	7.69	<0.01	0.03
Maine	1969	8	1	12.5	0.02	0.15
	1970	20a	ND			
Maryland	1969	12	ND			
	1970	19b	ND			
Massachusetts	1969	2	ND			
	1970	20a	ND			

Table 7.29 (continued)

State	Year	Number of sites analyzed	Number sites positive	Sites positive (%)	Mean residue level (ppm)	Maximum level detected (ppm)
Michigan	1969	51	1	2.0	<0.01	0.01
	1970	54	ND			
Minnesota	1969	NS				
	1970	120	2	1.67	<0.01	0.02
Mississippi	1960	29	1	3.5	0.01	0.19
	(endrin ketone)	29	1	3.5	<0.01	0.11
	1970	29	3	10.34	0.01	0.11
Missouri	1969	82	1	1.2	<0.01	0.01
	1970	81	1	1.23	<0.01	0.02
Nebraska	1969	106	1	0.9	<0.01	0.02
	1970	106	4	3.77	<0.01	0.03
Nevada	1969	2	ND			
	1970	NS				
New Hampshire	1969	2	ND			
	1970	20a	ND			
New Jersey	1969	5	ND			
	1970	19b	ND			
New Mexico	1969	10	ND			
	1970	NS				
New York	1969	38	1	2.6	0.01	0.56
	(endrin ketone)	38	1	2.6	<0.01	0.05
	1970	38	ND			

Table 7.29 (continued)

State	Year	Number of sites analyzed	Number sites positive	Sites positive (%)	Mean residue level (ppm)	Maximum level detected (ppm)
North Carolina	1969	31	2	6.5	<0.01	0.08
	1970	30	2	6.67	<0.01	0.03
North Dakota	1969	157	1	0.6	<0.01	0.01
	1970	NS				
Ohio	1969	68	ND			
	1970	69	1	1.45	<0.01	0.02
Oklahoma	1969	64	ND			
	1970	65	1	1.54	<0.01	0.06
Pennsylvania	1969	29	ND			
	1970	32	ND			
Rhode Island	1969	1	ND			
	1970	20a	ND			
South Carolina	1969	17	3	17.7	<0.01	0.05
	1970	17	ND			
South Dakota	1969	106	ND			
	1970	106	ND			
Tennessee	1969	27	1	3.7	<0.01	0.02
	1970	25	ND			
Utah	1969	12	ND			
	1970	NS				
Vermont	1969	5	ND			
	1970	20a	ND			

Table 7.29 (continued)

State	Year	Number of sites analyzed	Number sites positive	Sites positive (%)	Mean residue level (ppm)	Maximum level detected (ppm)
Virginia	1969	21	ND			
	1970	26c	ND			
Washington	1969	45	ND			
	1970	NS				
West Virginia	1969	6	ND			
	1970	26c	ND			
Wisconsin	1969	67	ND			
	1970	67	ND			
Wyoming	1969	17	ND			
	1970	NS				

ND = not detected; NS = not sampled in that year; a = 20 sites sampled in Maine, New Hampshire, Vermont, Massachusetts, Rhode Island, and Connecticut; b = 19 sites sampled in New Jersey, Maryland, and Delaware; c = 26 sites sampled in Virginia and West Virginia.

Source: modified from Crockett et al., 1971; Wiersma et al., 1972a.

Table 7.30. Summary of endrin residues in cropland soil by cropping region, FY 1969 and FY 1970

Crop	Sites showing detectable residues (%)		Mean concentration (ppm)	
	1969	1970	1969	1970
Corn	0.3	0.42	<0.01	<0.01
Cotton	7.3	5.94	0.01	<0.01
Cotton and general farming	2.6	4.27	<0.01	<0.01
General farming	3.6	1.36	<0.01	<0.01
Hay and general farming	ND	1.36	ND	ND
Irrigated land	11.1	2.56	0.01	0.01
Small grains	0.9	0.95	<0.01	<0.01
Vegetable	3.2	1.39	0.01	<0.01
Vegetable and fruit	6.1	4.76	0.01	0.01

ND = not detected.

Source: modified from Crockett et al., 1971; Wiersma et al., 1972a.

When soil samples were collected, an attempt was made to determine whether endrin had been used on the sites for the year of sampling. The locations at which endrin was admittedly applied are listed in Table 7.31. Endrin was applied to far fewer sites than the number at which it was detected. In 1969, 1684 sites were surveyed, but only 0.48% of these reported the use of endrin. In 1970, 0.45% of the 1346 sites surveyed had been treated with the pesticide. One explanation for the presence of endrin in nontreated soils is the persistence of residues from previous years of treatment. Under such circumstances, both the percentage of sites exhibiting contamination and the maximum residue level observed should decrease from year to year. One example is the state of Arkansas (Table 7.31), where endrin was applied in 1969 but not in 1970. Whereas 10.6% of the sites analyzed were positive for endrin in 1969, only 6.38% contained residues in 1970. The maximum levels detected for the two years were 0.29 and 0.03 ppm respectively.

The situation in Louisiana cannot, however, be explained in the same manner. In this case (Table 7.31), endrin was not applied in either year. Although the maximum detectable level decreased from 0.06 ppm in 1969 to 0.03 ppm in 1970, the percentage of samples exhibiting contamination more than doubled (3.7% in 1969 to 7.69% in 1970). Since the number of positive samples was small, this observation may not be statistically important. However, the possibility of other sources of contamination, such as soil drift, aerial fallout, and precipitation, must not be overlooked.

Table 7.31. States where endrin was used in FY 1969 and/or FY 1970

State	Year	Number of sites investigated	Number of sites treated with endrin	Sites treated with endrin (%)
Alabama	1969	23	2	8.70
	1970	22	1	4.55
Arkansas	1969	45	1	2.22
	1970	47	NU	
California	1969	66	NU	
	1970	43	1	2.33
Colorado	1969	60	3	5.00
	1970	NS		
Georgia	1969	28	NU	
	1970	30	1	3.33
Mississippi	1969	29	1	3.45
	1970	31	1	3.23
Nebraska	1969	103	NU	
	1970	106	2	1.89
North Dakota	1969	159	1	0.63
	1970	NS		

NU = no endrin used; NS = state not studied.

Source: modified from Crockett et al., 1971; Wiersma et al., 1972a.

The presence of endrin in cropland soil by crop in 1970 is presented in Table 7.32. During this year, endrin was applied only to cotton, and the highest percentage of contaminated sites as well as the highest levels of contamination were found in cotton fields. Once again, the occurrence of residues in fields where no endrin was applied suggests other means of contamination.

Monitoring for endrin in soils from the cotton-belt states was continued through 1973, and the data are presented in Table 7.33. Data for 1969 and 1970 are also included in this table so that trends may be easily observed (U.S. EPA, 1975c). Levels of endrin contamination reached their minimum values in Arkansas, Louisiana, and Mississippi in 1971. The trend, however, did not continue. Concentrations as high as 0.64 ppm were observed in Mississippi soils during 1972, while maximum concentrations in Arkansas and Louisiana soils during 1973 were 0.24 and 0.48 ppm respectively. No data are available as to the use of endrin in these states during 1972 and 1973.

Monitoring for endrin in soils planted to specific types of crops other than cotton has also been reported. Fields in the eastern states planted to root crops were surveyed in the fall of 1965 (Seal et al., 1967).

Table 7.32. Endrin in cropland soil by crop — FY 1970

Crop	Number of sites sampled	Number of times detected	Occurrence (%)	Mean concentration (ppm)	Maximum concentration (ppm)
Alfalfa and bur cloves	114	3	2.63	<0.01	0.07
Field corn	363	4	1.10	<0.01	0.03
Cotton	49	7	14.29	0.01	0.16
Grass and hay	29	ND			
Mixed hay	118	ND			
Soybeans	254	2	0.79	<0.01	0.03

ND = not detected.

Source: modified from Crockett et al., 1971.

Table 7.33. Endrin in cotton-belt soils: 1969–1973

State	Year	Number of sites analyzed	Number of sites positive	Sites positive (%)	Mean residue level (ppm)	Maximum level detected (ppm)
Alabama	1969	22	2	9.1	<0.01	0.05
	1970	12	2	9.5	0.01	0.16
	1971	0	NA	NA	NA	NA
	1972	23	1	4.3	0.02	0.42
	1973	22	1	4.5	<0.01	0.10
Arkansas	1969	47	5	10.6	0.01	0.29
	1970	47	3	6.4	<0.01	0.03
	1971	22	NA	NA	NA	NA
	1972	46	1	2.2	<0.01	0.10
	1973	43	2	4.7	0.01	0.24
Louisiana	1969	27	1	3.7	<0.01	0.06
	1970	26	2	7.7	<0.01	0.03
	1971	22	ND	NA	NA	NA
	1972	26	ND	NA	NA	NA
	1973	27	1	3.7	0.02	0.48
Mississippi	1969	29	1	3.4	0.01	0.19
	1970	29	3	10.3	0.01	0.11
	1971	38	1	2.6	<0.01	0.01
	1972	31	2	6.5	0.02	0.64
	1973	30	ND	NA	NA	NA
Texas	1971	113	1	0.8	<0.01	0.05

Source: modified from U.S. EPA, 1975c.

Endrin was detected in 10 out of 49 fields sampled. Eight of the fields were planted with potatoes, and the concentration of endrin ranged from 0.08 to 0.50 ppm (average was 0.27 ppm). The remaining two fields were planted with carrots. The range of endrin concentrations was 0.05 to 0.10 ppm (average was 0.08 ppm). Endrin residues were not detected in either crop.

Commercially grown onions and the soils on which they were grown were sampled in the ten major onion-producing states in 1969 and analyzed for the presence of endrin (Wiersma et al., 1972c). Endrin was detected in 15.5% of the 76 sites sampled at an average concentration of 0.06 ppm (range was 0.01 to 2.05 ppm). Residues were absent from onions per se. Soil from commercial wheat fields was sampled in the 16 major wheat-producing states during the summer of 1969 (Wiersma et al., 1971a). Endrin had been applied at the rate of 0.33 lb/acre to four of the 100 wheat fields sampled. Endrin was detected at each of these four sites at an average concentration of 0.03 ppm (range was 0.02 to 0.08 ppm), but was not detected at any other site. No endrin was detected in the harvested wheat crops.

The NSMP sampled several urban areas annually for pesticide residues. The results of the 1969 to 1973 surveys on endrin are present in Table 7.34 (U.S. EPA, 1975c; Wiersma et al., 1972a). From the limited data available, accumulation of endin residues does not appear to present a major problem to urban environments.

Monitoring for pesticide residues in soils from agricultural regions in Canada has also been conducted. Southwestern Ontario is an area of intensive farming with a wide variety of soil types and crops. The cyclodiene insecticides were utilized to a considerable extent between 1954 and 1960 and to a lesser extent in subsequent years. In 1964, soil samples were collected from 31 farms throughout the region and analyzed for the presence of organochlorine insecticides (Harris et al., 1966). Fifty percent (16) of the samples contained significant amounts of cyclodiene insecticides (greater than or equal to 0.1 ppm). However, only eight of the samples were active enough to cause mortality in appropriate test organisms. Endrin was found at a concentration of 3.8 ppm at one site where the pesticide had been applied annually since 1960. Trace amounts (less than 0.1 ppm) were detected at two other sites where endrin had been surface applied either in 1963 or in 1964. Endrin was neither used nor detected at any other site surveyed that year. The study was continued through 1969 on 16 of the original 31 farms (Table 7.35) (Harris and Sans, 1971). In the 1964 survey, endrin had been found on only two of these farms; by 1966, residues were present on seven farms, and by 1969, detectable levels were reported on nine farms. Concentration ranges were 0.11 to 3.76 ppm in 1964, 0.01 to 6.55 ppm in 1966, and 0.01 to 3.54 ppm in 1969. Although endrin was reportedly applied to only 3 of the 16 farms sampled, more extensive use during this period was probable. Endrin was a likely substitute for certain other pesticides phased out between 1958 and 1969 due to development of resistant strains or to bans. Endrin usage was apparently most extensive in tobacco fields, but the highest residue levels were present in soils where vegetables had been grown. These soils

Table 7.34. Endrin residues in urban soils 1969–1973

Urban area	No. of sites	No. of positive detections	Positive detections (%)	Mean (ppm)	Highest level detected (ppm)
		1969			
Bakersfield, CA	50	3	6.0	0.01	0.03
Camden, NJ	50	ND			
Houston, TX	50	1	2.0	0.01	0.01
Manhattan, KS	50	ND			
Miami, FL	50	ND			
Milwaukee, WI	50	ND			
Salt Lake City, UT	50	1	2.0	0.01	0.06
Waterbury, CT	50	ND			
		1970			
Augusta, ME	27	ND			
Charleston, SC	27	ND			
Cheyenne, WY	19	ND			
Grand Rapids, MI	23	ND			
Greenville, MS	28	ND			
Honolulu, HI	21	ND			
Memphis, TN	28	1	3.6	0.01	0.07
Mobile, AL	29	ND			
Portland, OR	25	ND			
Philadelphia, PA	26	ND			
Richmond, VA	27	ND			
Sikeston, MO	27	ND			
Sioux City, LA	22	ND			
Wilmington, DE	27	ND			
		1971			
Baltimore, MD	156	ND			
Gadsen, AL	55	ND			
Hartford, CT	48	ND			
Macon, GA	43	1	2.3	0.01	0.17
Newport News, VA	78	ND			
		1972			
Des Moines, IA	82	ND			
Fitchburg, MA	35	ND			
Lake Charles, LA	70	ND			
Pittsburgh, PA	189	ND			
Reading, PA	49	ND			

Table 7.34 (continued)

Urban area	No. of sites	No. of positive detections	Positive detections (%)	Mean (ppm)	Highest level detected (ppm)
		1973			
Evansville, IN	82	ND			
Greenville, SC	86	ND			
Pittsvield, MA	45	ND			
Tacoma, WA	95	ND			
Washington, DC	116	ND			

ND = not detected.

Source: modified from U.S. EPA, 1975c.

Table 7.35. Residues of endrin found in agricultural soils on 16 farms in southwestern Ontario in 1964, 1966, and 1969

Site number	Endrin residues in soil (ppm)				Endrin applied (year)
	General classification	1964	1966	1969	
1	Field	ND	0.01	0.01	
2	Field	ND	ND	0.10	
3	Field	ND	ND	ND	
4	Field	ND	ND	ND	
5	Tobacco	0.11	0.10	0.06	1966, 1969
6	Tobacco	ND	0.10	0.07	
7	Tobacco	ND	0.03	0.03	
8	Tobacco	ND	0.05	0.04	1964
9	Vegetable	ND	ND	NS	
10	Vegetable	ND	0.38	0.08	
11	Vegetable	3.76	6.55	3.54	1960–1964
12	Vegetable	ND	ND	ND	
13	Vegetable	ND	ND	ND	
14	Vegetable	ND	ND	1.06	
15	Fruit	ND	ND	ND	
16	Fruit	ND	ND	ND	

ND = not detected (<0.01 ppm in 1964; <0.001 ppm in 1966 and 1969); NS = not sampled in that year.

Source: modified from Harris and Sans, 1971.

were classified primarily as "muck" and, as such, contained a large quantity of organic matter to which the insecticides readily adsorbed. Since only 0.13% of the total area of the Province of Ontario is devoted to nonfield crop farming (vegetable, tobacco, and orchard), where insecticides are used extensively, contaminated soils are expected to be concentrated in relatively small pockets and are not representative of the entire province.

The levels of organochlorine insecticide residues in soil and legume crops in the northeastern agricultural area of Saskatchewan, Canada, were studied in September 1966 (Saha et al., 1968). Soil and foliage samples were taken from 16 sweet clover and 4 alfalfa fields. Fifteen percent of the soil samples contained endrin residues at concentrations ranging from 0.01 to 0.02 ppm. Residues of endrin in the crops were either undetectable or present in only trace amounts (less than 0.01 ppm).

Saha and Sumner (1970) analyzed 41 agricultural soil samples from 21 vegetable farms in Saskatchewan for the presence of organochlorine insecticide residues. Aldrin and/or dieldrin were present in 61% of the samples; endrin was detected only once at a concentration of 0.48 ppm.

Agricultural soil from Japan was analyzed for organochlorine insecticide residues in a study by Suzuki et al. (1973a). Endrin was detected in 29 out of 99 samples tested. The concentration range was 0.016 to 0.629 ppm with a mean of 0.183 ppm.

7.5.4 Persistence of Endrin in the Lithosphere

Many pathways have been established for the removal of endrin from the soil. Mechanisms such as volatilization, leaching into groundwater, wind erosion, and surface runoff result in the translocation of endrin to the atmosphere or hydrosphere and were discussed in detail in the sections dealing with sources of endrin in those compartments (Sect. 7.3.2 and 7.4.2). Other pathways, such as direct root uptake by plants and ingestion by soil invertebrates, lead to contamination of living things and are also dealt with in the appropriate sections (4. and 5.6). The only remaining pathways for the removal of endrin from the soil are photodecomposition, thermal decomposition, and bacterial degradation. Although the first two were discussed in Sect. 2.3 and the third in Sect. 3.2.1, the fact that these pathways comprise the only mechanism for actual dissipation of endrin from the environment (as opposed to mere translocation) deems it appropriate that they be briefly discussed in this section as well.

7.5.4.1 Mechanisms for Removal of Endrin from the Lithosphere

7.5.4.1.1 Photodecomposition and thermal decomposition. It has been known for some years that endrin undergoes chemical isomerization under the influence of sunlight and heat (Klein and Korte, 1967). Most of the laboratory studies dealing with the photo- and thermochemistry of endrin have been conducted either in the solid state or in solution in an organic solvent. The results of these studies appear most relevant to endrin in the lithosphere, since the pesticide may reside there either as a solid or adsorbed to the organic matter in the soil.

The extent to which endrin may be expected to disappear as a result of the light and heat received from sunlight was studied by Baker and Applegate (1974). Samples of solid endrin were placed in a petri dish and exposed to ultraviolet radiation in the biologically active range (300 to 400 nm) for from 4 to 24 hr. Half of the samples were covered with aluminum foil in order to separate the effects of the uv rays from those of heat (35°C). Samples were analyzed for endrin at four intervals by electron-capture gas chromatography; the results are presented in Table 7.36.

After 24 hr of uv exposure, 52.4% of the primary gas-chromatographic endrin peak had been lost. In the presence of heat alone, the losses amounted to only 20.1%. The net effect of uv radiation on the primary peak was, therefore, 32.3% over the 24-hr period. The effects of uv radiation on the secondary peak were similar to those for the primary peak. Ultraviolet exposure resulted in the loss of 59.4% of the secondary peak after 24 hr, while heat alone diminished the peak by only 29.9%. The net uv decomposition as indicated by the secondary peak was 29.5%. Since decomposition products were neither identified nor quantitated, it is not known whether exposure to heat resulted in thermal isomerization, vaporization, or both. The experiment only indicates that under these conditions of simulated sunlight, uv decomposition does, in fact, occur.

The major products resulting from the photochemical and thermochemical isomerization of endrin have been identified, and their structures, along with that of the parent compound, are presented in Sect. 2.

7.5.4.1.2 Degradation by soil microorganisms. The removal of persistent pesticides from soil may be accomplished by microbial degradation, providing that an appropriate microbial species is present and that the soil conditions are suitable.

Matsumura et al. (1971) screened about 150 microbial isolates from various soil samples for their ability to degrade ^{14}C-labeled endrin. Twenty-five of these organisms were active in degrading endrin, but the degree to which degradation occurred and the number of products varied considerably (Sect. 3.).

In a similar study (Patil et al., 1970), 20 microorganisms, which had previously been shown to degrade dieldrin, were tested for their ability to degrade endrin. All were positive; however, no degradation products were described (Sect. 3.).

The importance of such factors as soil pH, moisture content, the presence or absence of crops, and soil depth in determining whether or not microbial degradation of endrin is probable was studied by Nash et al. (1972). In one experiment, Lakeland sandy loam, pH 6.4, was treated with 20 ppm ^{14}C-labeled endrin at one of three soil depths (1 to 2 cm, 16 to 17 cm, or 31 to 32 cm). A soybean plant was grown in the soil for 75 days, after which time the 1-cm treated sections were extracted and analyzed for endrin and its degradation products by both gas and thin-layer

Table 7.36. Effect of ultraviolet radiation on the loss of endrin from petri dishes treated with 100 μg of sample

	Quantity remaining after the given number of hours (μg)					
	0	4	8	16	20	24
Primary peak (+uv)	100	87.2 ± 1.2	77.9 ± 5.1	65.4 ± 2.4	51.1 ± 1.3	47.6 ± 1.3
Primary peak (−uv)	100	96.2 ± 1.4	89.8 ± 2.1	86.8 ± 1.5	82.1 ± 2.3	79.9 ± 3.9
Secondary peak (+uv)	100	78.3 ± 3.8	71.8 ± 1.2	60.8 ± 1.6	44.4 ± 2.8	40.6 ± 1.1
Secondary peak (−uv)	100	88.0 ± 1.3	84.5 ± 1.6	82.1 ± 1.1	75.9 ± 1.9	70.1 ± 2.2

Source: R. D. Baker and H. G. Applegate, *Tex. J. Sci.* 25(1-5): 53-59 (1974). Copyright 1974 Texas Academy of Science.

chromatography. At least three and possibly five different soil transformation products were detected. Endrin ketone and endrin alcohol were positively confirmed by both analytical techniques; endrin aldehyde was tentatively confirmed. Two other polar degradation products remained unidentified. The amount of endrin and its three major transformation products found in the treated soil layers are given in Table 7.37. Considerable transformation occurred at all depths, with endrin ketone being the primary product. Ratios of the three products to recovered endrin indicate that transformation increased with depth, suggesting that the responsible organisms require anaerobic conditions.

In another experiment by the same group, two uncultivated samples of (1) the pH 6.4 Lakeland sandy loam and (2) a more acid variety of the same soil (pH 4.2 Lakeland sandy loam) were sprayed with endrin. One sample of each had been kept in an air-dried state, while the other had been moistened with a water spray. Following a 75-day incubation, all four samples were extracted and analyzed for endrin and its transformation products; the results are presented in Table 7.38.

In the absence of the soybean plant, no transformation was detected in the pH 6.4 soil in either the dry or moist state. With the more acidic soil, some transformation occurred following treatment of the dry soil only. The major product was endrin ketone, but a second product, tentatively identified as endrin aldehyde, was also detected.

Erroneous conclusions as to the probable persistence of endrin in the soil may result if the fate of the pesticide is studied in only one type of soil under a single set of conditions. Bartha et al. (1967) studied the stability of endrin and its effect upon CO_2 production and nitrification in Nixon sandy-loam soil, pH 6.0. Soil samples supplemented with $NH_4H_2PO_4$ and containing either 250 to 2500 ppm endrin were brought to 70% of moisture holding capacity and incubated at 28°C for 30 days. Carbon dioxide production was measured at intervals throughout the duration of the experiment. Microbial degradation of the endrin would have resulted in an increased CO_2 production relative to that of an identical soil sample in the absence of endrin. No such increase occurred. On the contrary, a 20% decrease in CO_2 production was observed at both endrin concentrations, indicating that the pesticide was toxic to some of the soil microorganisms. Addition of glucose reversed this inhibition of respiration and brought CO_2 production back to 100% of the control levels. Endrin, at a concentration of 250 ppm, had no effect on nitrate production in this soil under similar conditions. The higher concentration (2500 ppm) was not studied for effects upon nitrification. From these experiments it might be concluded that endrin is not degraded by the microbial population of Nixon sandy-loam soil. However, as was observed in the study by Nash et al. (1972), actual field conditions and the presence of crops may alter the circumstances sufficiently to allow degradation to take place.

7.5.4.2 Factors affecting the persistence of endrin in the lithosphere. The extent to which any of the pathways succeed for removal of endrin from the soil depends, in turn, upon the interplay of a large

Table 7.37. Influence of treatment depth and one soybean crop on endrin residues in Lakeland sandy loam (pH = 6.4)

Soil depth (cm)	Compound							Total products (ratio[b])	Total residues	
	Endrin (%)	Endrin alcohol		Endrin aldehyde[a]		Endrin ketone				
		%	Ratio[b]	%	Ratio[b]	%	Ratio[b]		g/g	%
1–2	57.3	7.8	0.13	9.4	0.16	27.7	0.48	0.78	1.856	79.4
16–17	42.1	8.4	0.20	14.1	0.33	35.5	0.84	1.38	1.018	
31–32	35.3	8.2	0.23	16.0	0.45	40.5	1.15	1.83	1.849	

[a] Tentatively identified.

[b] Ratio of transformation product to endrin.

Source: R. G. Nash, M. L. Beall, Jr., and W. G. Harris, *J. Environ. Qual.* 1(4): 391-394 (1974). Copyright 1974 American Society of Agronomy.

Table 7.38. Influence of soil moisture on endrin treatment of Lakeland sandy loam

Soil pH	Compounds found				
	Soil moisture	Endrin	Endrin alcohol	Endrin ketone	Endrin aldehyde[a]
4.2	Dry	+[b]	–[c]	+	+
4.2	Moist	+	–	–	–
6.4	Dry	+	–	–	–
6.4	Moist	+	–	–	–

[a] Tentatively identified.

[b] Positive.

[c] Not detected.

Source: R. G. Nash, M. L. Beall, Jr., and W. G. Harris, *J. Environ. Qual.* 1(4): 391-394 (1974). Copyright 1974 American Society of Agronomy.

number of influential parameters. Included among these are the physical and chemical properties of the pesticide, physical and chemical properties of the soil, microbial population of the soil, type of crops planted, agricultural practices employed, topography, temperature, humidity, amount of rainfall, irrigation and dew, average wind velocity, and intensity of sunlight. Since many of these parameters affect more than one pathway, it is only possible to evaluate the effect of each parameter upon persistence per se. To estimate the net result of the combined effects of all parameters upon all pathways under natural conditions is, however, exceedingly difficult. An understanding of the direction and extent of a given parameter upon persistence can, therefore, only serve as a guideline for assessing the probability that the pesticide in question will persist under a given set of conditions.

7.5.4.2.1 Soil organic content. Due to their hydrophobic, lipophilic nature, organochlorine insecticides are expected to adsorb to the organic matter present in soil. It follows that a correlation should exist between persistence of these insecticides in various soils and the percent organic matter in those soils. The difficulties of establishing such correlations are revealed in the laboratory studies of Bowman et al., 1965. The adherence of endrin to various soils was examined by determining the extent to which the pesticide could be removed from soil columns by percolation with either hexane or distilled water. Endrin was applied to the top 2 cm of the columns, and the soils were eluted at atmospheric pressure. Fractions were extracted, pooled, and analyzed by electron-capture gas chromatography; the results are summarized in Table 7.39. Endrin remained totally adsorbed to three of the eight soils tested and moderately adsorbed to two others following elution with hexane. No relationship existed between the percent organic matter in these soils and the amount of endrin eluted. However, the range of soil organic content among these soils was narrow (0.17 to 1.33%) and might not have been sufficient for a statistically significant comparison.

The Rutledge sands I and II containing 6.56 and 19.43% organic matter, respectively, exhibited the least retention of endrin, releasing virtually all of the bound pesticide to hexane with the first 100 ml of eluate. The unexpected behavior of these soils, which responded in a similar fashion toward a variety of other organochlorine insecticide as well, was most probably related to some property other than percent organic content. One other soil, Greenville sandy clay, also released 98% of the bound endrin to hexane. In this case, however, the behavior was not inconsistent with soil organic content (0.57%).

The Rutledge sands behaved as expected when an attempt was made to elute the adsorbed endrin with distilled water. No trace of endrin was detected following elution with 1600 ml of water. Difficulties in relating this behavior to organic content arise, however, when it is considered that Magnolia sandy-clay loam (organic content 0.72%) and Magnolia sandy loam (organic content 1.33%) also retained the major portion of the applied endrin under the same elution conditions. Trace amounts of endrin degradation products were detected in the aqueous eluates from Magnolia

Table 7.39. Elution characteristics of endrin on various types of soil as determined by percolation with hexane and distilled water

Soil	Organic matter (%)	Hexane Eluate fraction (ml)	Hexane Added endrin in eluate (%)	H_2O Eluate fraction (ml)	H_2O Added endrin in eluate (%)	Endrin aldehyde in eluate (%)	Endrin ketone in eluate (%)	Unidentified degradation product (%)
Lynchburg loamy sand	0.17	A[a]	0 } 0	D[d]	0 } 0	3 } 5	53 } 56	T } 1
		B[b]	0	E[e]	0	2	3	1
		C[c]	0					
Lakeland sand deep phase	0.42	A	10 } 18	D	52 } 72	T } T	6 } 6	1 } 1
		B	4	E	20	0	0	0
		C	4					
Greenville sandy clay	0.57	A	98 } 98	D	15 } 51	0 } 0	T } T	0 } 0
		B	0	E	36	0	T	0
		C	0					
Magnolia sandy-clay loam	0.72	A	5 } 26	D	0 } 0	0 } 0	T } T	0 } 0
		B	21	E	0	0	0	0
		C	T					
Susquehanna sandy clay	0.77	A	0 } 0	D	20 } 95	0 } 0	T } T	0 } 0
		B	0	E	75	0	0	0
		C	0					
Magnolia sandy loam	1.33	A	0 } 0	D	0 } 0	0 } 0	0 } T	0 } 0
		B	0	E	0	0	T	0
		C	0					
Rutledge sand I	6.56	A	98 } 98	D	0 } 0	0 } 0	0 } 0	0 } 0
		B	T[f]	E	0	0	0	0
		C	0					
Rutledge sand II	19.43	A	104 } 104	D	0 } 0	0 } 0	0 } 0	0 } 0
		B	T[f]	E	0	0	0	0
		C	0					

[a] A = 0 to 100 ml.
[b] B = 100 to 300 ml.
[c] C = 300 to 500 ml.
[d] D = 0 to 800 ml.
[e] E = 800 to 1600 ml.
[f] T = Trace (<0.5%).

Source: modified from Bowman et al., 1965.

sandy loam, Magnolia sandy-clay loam, Greenville sandy clay, and Susquehanna sandy clay. Seven percent of the endrin residues eluted from Lakeland sand (deep phase) and 62% of those eluted from Lynchburg loamy sand consisted of degradation products. The major degradation product observed in all cases was endrin ketone. However, endrin aldehyde and another unidentified product were also detected. No degradation occurred in the Rutledge sands, but whether the lack of degradation is related to the high organic content of these soils is uncertain.

In a study by Gish (1970), agricultural soils were collected from 67 fields in 8 states of the southern and midwestern United States and analyzed for the presence of endrin. Since the soil types and organic contents were also reported, it was possible to examine the data for correlation between endrin persistence and soil organic content. Sixteen of the 67 sites surveyed contained endrin residues. The range was from less than 0.005 to 3.47 ppm, with a median of 0.051 ppm and an arithmetic mean of 0.49 ppm. Once again, no relationship was noted between the percent organic content of the soil and the degree to which endrin persisted The highest endrin concentration (3.47 ppm) was, in fact, detected in the soil having the lowest organic content (2.98%). However, as in the case of the Bowman study, the range of organic content was also rather small (2.98 to 6.70%) and, under the complex conditions which exist naturally in the field, may not allow for realistic correlation.

Evidence that organic content of soil may indeed influence persistence can be found in the data of Harris and Sans (1969). Three experimental plots, set up in 1967 at different locations throughout southwestern Ontario, were sampled for organochlorine insecticide. Although the pesticide use records for these plots were not revealed, it was believed that all had received reasonably similar treatments in previous years. The soils were sandy loam, clay, and muck and contained 1.4, 3.6, and 66.5% organic matter respectively. The concentrations of total cyclodiene residues for these soils were 0.76 ppm for the sandy loam, 1.34 ppm for the clay, and 10.44 ppm for the muck. Although endrin concentrations were higher in the sandy loam (0.14 ppm) than in the clay, the highest residues observed were in the highly organic muck soil (5.94 ppm). It therefore seems probable that the quantity of organic matter present in soil does influence persistence but that a significant effect is observed only when the percentage of organic matter is large.

7.5.4.2.2 Moisture and mixing. Since microbial degradation of organochlorine insecticide occurs predominantly under anaerobic conditions, little loss of surface-applied pesticide can be expected to occur by this route. In an attempt to accelerate the disappearance of six chlorinated insecticides from the top 10 cm of soil, the effects of alternately wetting and drying the soil and of mixing the soil were examined (Guenzi et al., 1971). It was believed that the former practice would accelerate volatilization, codistillation, and leaching, while the latter would continuously expose new residues to the surface layer for volatilization and possible thermal degradation. Experimental plots of Nunn silty-clay loam were set up at the Colorado State University farm. Each of the pesticides to be studied was added to the top 10 cm of soil to a final concentration of 25 ppm. After application, soils were treated in one of two ways: (1) 2.5 cm of water was added every week, or (2) 2.5 cm of water was added every week and the surface 8 cm of soil was mixed every two weeks. For each of these sets of treatments, one plot received no added energy source, while a second received 0.25% alfalfa mixed in the top 10 cm of soil. Soils were sampled and analyzed for the presence of the added pesticide immediately after pesticide applications and at given intervals thereafter for a period of 22 weeks, after which all mixing and water treatments were discontinued, and the plots received only natural precipitation. The soils were sampled once more for distribution and recovery of pesticides one year following the initiation of the experiment. All analyses were by electron-capture gas chromatography; the results for endrin are presented in Table 7.40.

Table 7.40. Recovery of endrin from soil at various depths after one year; initial concentration was 25 ppm

Depth (cm)	W and D[a]				W and D + M[b]			
	OA[c]		A[d]		OA		A	
	ppm	%	ppm	%	ppm	%	ppm	%
0-16	12.05	85.5	12.73	89.0	11.63	82.0	12.68	88.0
16-32	0.60	3.2	1.30	7.1	1.70	3.7	0.72	3.7
32-48	0.13	0.7	0.18	0.8	0.10	0.4	0.10	0.4
Total		89.4		96.9		86.1		92.1

[a] 2.5 cm of water added every week and no soil mixing for 22 weeks — then discontinued.

[b] 2.5 cm of water added every week and the surface 7.6 cm of soil mixed every 2 weeks for 22 weeks — then discontinued.

[c] No added energy source.

[d] 0.25% alfalfa mixed in the top 10 cm of soil.

Source: W. D. Guenzi, W. E. Beard, and F. G. Viets, Jr., *Soil Sci. Soc. Am. Proc.* 35(6): 910-913 (1971).

No differences in endrin concentration in the 0 to 8 cm of soil layer were observed among the variously treated experimental plots over the 22-week period. The mean concentration during the 22-week period remained at 20 ppm or 80% of the initial amount. The low recovery (expected 95 to 100%) was attributed to a small vertical distribution of the endrin as a result of the mechanics of adding and packing and amended soil. In addition, some of the pesticide may have remained in the emulsion form in which it was applied and been leached to a small extent during the first water addition.

The total recovery of endrin one year following the initiation of the study (Table 7.40) was also approximately the same for each of the experimental plots (91%). The endrin was, however, found to be distributed throughout the 48 cm of soil depth. Most of the pesticide was found in the 0- to 16-cm layer, and about 4% and less than 1% were detected in the 16- to 32-cm layer and the 32- to 48-cm layer respectively.

It is interesting to note that concentrations of some other pesticides, namely, lindane and heptachlor, were greatly affected by similar treatments. After 22 weeks, only 54% of the lindane was recovered from the top 8-cm layer following alternate wetting and drying, while only 44% remained when mixing was also performed. Forty-three percent of the added heptachlor remained after 22 weeks of alternate wetting and drying, while mixing reduced this value to 27%. Alfalfa amendment had no influence upon the persistence of either pesticide. One year later, 82% of the lindane was recovered from the combined 48-cm soil plot which had been subjected to wetting and drying only, while 70% was recovered from the mixed soil. The disappearance of lindane observed during the initial 22 weeks was therefore due primarily to leaching into the lower soil strata, although some volatilization did occur. Loss of heptachlor, on the other hand, was predominantly through volatilization, since only 57% was recovered from the undisturbed soil and 42% from the mixed soil after one year. From these studies it would be predicted that decontamination of soil containing lindane and heptachlor could be accelerated by tilling the soil. This process would, however, have little effect on reducing residues of endrin under similar conditions.

7.5.4.2.3 Flooding. Microbial degradation of persistent pesticides may be accelerated by practices leading to anaerobic soil conditions. Flooding the soil is one method by which anaerobiosis may be stimulated. In addition, amendment of the soil with a supplementary microbial energy source such as alfalfa assists the process. The readily available energy source allows the aerobic soil microorganisms to rapidly deplete the available oxygen, thereby creating a suitable environment for the anaerobic degradation of the pesticides (Guenzi et al., 1971) (Sect. 3.).

Castro and Yoshida (1971) studied the degradation of endrin in soils from the Philippines under both flooded and upland conditions. Four types of soil were tested, and their characteristics are presented in Table 7.41.

Endrin proved unstable only in the flooded Casiguron soil. After two months, only 8.4% of the pesticide was recovered from the flooded soil as compared with 88.24% from the upland soil. Endrin persisted in all of the other soils regardless of flooding. Casiguron soil contained a higher percentage of organic matter (4.4%) than any of the other three soils studied.

Table 7.41. Soils tested for their ability to degrade endrin

Soil type	Organic matter (%)	Total nitrogen (%)	pH
Maahas clay	2.0	0.14	6.6
Luisiana clay	3.2	0.21	4.7
Pila clay loam	1.5	0.09	7.6
Casiguran sandy loam	4.4	0.2	4.8

Source: modified from Castro and Yoshida, 1971.

Studies to determine the persistence of endrin in flooded vs nonflooded soils indicated endrin to be degraded more rapidly under anaerobic (flooded) conditions. Aerobic soil conditions increased persistence of endrin in soil. The addition of rice straw to the water enhanced endrin's degradation. Data also indicated microbial participation in the decomposition process (Gowda and Sethunathan, 1976; Gowda and Sethunathan, 1977).

7.5.4.2.4 Crop rotation. Some evidence exists that crop rotation may aid in the dissipation of endrin from the soil. Endrin at the rate of 1.6 lb of active ingredient per acre was applied to two sets of dry-land plots (Pullman clay loam soil) in June 1959 (Daniels, 1966). One set was continuously cropped for 11 years with grain sorghum, while the other set was a rotation of grain sorghum, fallow, and wheat. The plots were cultivated normally. Soils were analyzed 5 and 11 years after pesticide application for the presence of endrin; the results are presented in Table 7.42. Less endrin residues were found in the rotation plots than in the continuously cultivated plots, and concentrations were generally

Table 7.42. Endrin residues found in Pullman clay loam soil 5 and 11 years after application

Crop	Residues at indicated soil depth (ppm)			
	Upper 6 in.		Lower 6 in.	
	5 years	11 years	5 years	11 years
Continuous sorghum	0.069	0.02	0.009	<0.0009
Sorghum-fallow-wheat rotation	0.029	<0.0009	<0.003	0

Source: modified from Daniels, 1966.

greater in the first 6 in. than in the lower 6 in., indicating that movement into lower soil strata is minimal. Even though the endrin concentration was considerably diminished, the remaining residues continued to provide satisfactory control of wireworms and grubs for 11 years after treatment.

The type of crop used for rotation and the subsequent treatment of the crop undoubtedly affect persistence under field conditions. Rye, for instance, is usually ploughed down in order to build up the soil and will not help remove endrin when rotated with an endrin-treated crop such as tobacco. Rotation of tobacco with winter wheat to corn will help in removal of endrin from the soil, since these crops will absorb the pesticide prior to harvest (Harris et al., 1966).

7.5.4.2.5 Type of endrin formulation. Endrin formulations are prepared as dusts, wettable powders, emulsions, and sprays. The type of formulation may affect the persistence of the pesticide in the soil. Sprays tend to disappear sooner than dusts due to a faster initial volatilization. However, loss from drift is more significant in the case of dusts (Carey et al., 1973).

The mineral clays used as diluents for the dust and wettable powder formulations have been shown to catalyze the decomposition of endrin (Fowkes et al., 1960). Investigations showed that the decomposition was promoted by the acid sites on the clay and that appropriate neutralization could reduce decomposition to negligible rates. Since the introduction and widespread use of clay deactivators, the probability of decomposition by this mechanism is virtually zero.

7.5.4.3 Studies on the persistence of endrin in the lithosphere. Several studies have been performed to determine the long-term persistence and rates of disappearance of endrin in the soil. In 1951, duplicate soil plots of Congaree sandy-loam soil were established at the Plant Industry Station, Beltsville, Maryland (Nash and Woolson, 1967). Endrin was applied to the soil at rates of 56 and 224 kg/ha (25 and 100 ppm), and the soils were thoroughly mixed prior to placement in the 38-cm plots. The soils were cropped at various times through 1962, after which time cultivation was discontinued and weeds were cut when necessary. Soil samples were taken at intervals through 1966 and analyzed for the persistence of endrin residues either by estimation of total chlorine content (through 1962) or by electron-capture gas chromatography. The decline of endrin in Congaree sandy-loam soil is presented in Fig. 7.10. The quantity of endrin remaining in the soil 14 years after application was 41% of the applied amount, and the calculated half-life under the experimental conditions was approximately 11.8 years. No difference in persistence was noted between the higher and lower rates of application.

In 1967, the plots were sampled again and degradation products were identified (Nash and Harris, 1973). The total remaining residues were 39.2% of the originally applied amount. Of the remaining residues, 54.8% was endrin itself, 38.4% was endrin ketone, 5.7% was tentatively identified as endrin aldehyde, and 1.1% was endrin alcohol.

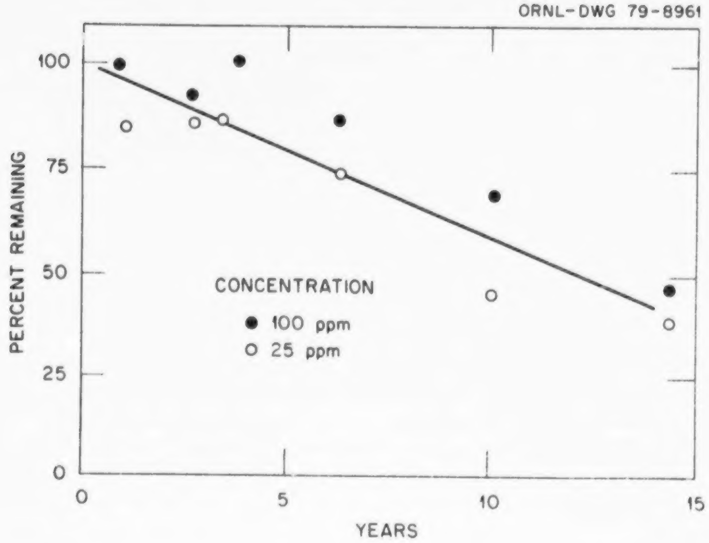

Fig. 7.10. Persistence of endrin in Congaree sandy-loam soil. Source: R. G. Nash and E. A. Woolson, *Science* 157: 924-927 (1967). Copyright 1967 by the American Association for the Advancement of Science.

Due to the manner in which these experimental plots were established and maintained, an upper limit of soil persistence would be expected (Nash and Woolson, 1967). Photodecomposition, which occurs only near the surface of the soil, was probably minimal due to the initial thorough mixing of endrin throughout the soil and absence of tillage. Volatilization, which occurs most rapidly at the soil surface, would also have been depressed by the same procedures, although some losses may have occurred following upward diffusion of the pesticide or possibly through codistillation with water. The extremely high rates of endrin application may have eliminated much of the soil's zoological and microbial populations, thereby reducing both microtillage and microbial degradation. Leaching was minimized by the placement of a gravel layer beneath the soil column. Mechanical removal via absorption by crops and subsequent harvest was also negligible, since these plots were not cultivated as extensively as normal agricultural soils. Thus, endrin is expected to be somewhat less persistent in the same soil under natural field conditions.

The degree to which endrin accumulates in the soil following repeated foliar applications over the course of two to three years was studied by Foster et al. (1956). Experimental plots of Congaree sandy loam, Koufman sandy loam, and Sassofras sandy loam were set up at Beltsville, Maryland; State College, Mississippi; and New Brunswick, New Jersey respectively. Due to differences in climate and soil among the three locations, the test crops grown differed widely. Successions of crops were planted, sprayed with endrin (in the form of a 50% wettable powder), and harvested over a two- to three-year period from 1951 to 1954. The cumulative amount of endrin applied at each location and the percent remaining in the soil at the end of the experiment are presented in Table 7.43. Since all

Table 7.43. Accumulation of endrin in soil following repeated foliar application

Location	Soil type	Years of study	Total amount applied (lb)	Amount remaining[a] (%)
Beltsville, MD	Congaree sandy loam	1951–1954	65	78
State College, MS	Kaufman sandy loam	1951–1953	35	25
New Brunswick, NJ	Sassafras sandy loam	1952–1954	30	32

[a] Percentage of amount applied.

Source: modified from Foster et al., 1956.

analyses were for total organic chlorine in the soil, they include degradation products as well as endrin itself. The percent accumulation of endrin in the State College and New Brunswick soils was strikingly less than that observed at Beltsville. It is not known to what extent these differences were caused by differences in the soils, the crops, the climate, or the cultivation practices. But these results do indicate the inadvisability of predicting how much endrin will accumulate in an area of a given soil on the basis of studies conducted at a different location with other types of soil.

The distribution of endrin throughout the Congaree sandy-loam plots at Beltsville 13 years following the last foliar application was studied by Nash and Woolson (1968). Soil cores from each plot were taken and sectioned into 7.6-cm lengths. The soils were extracted and the extracts analyzed for endrin by gas chromatography; the results are presented in Fig. 7.11. Approximately 80% of the residual endrin was found in the upper 23 cm of soil, which probably corresponds to the cultivated layer. Due to the gravel layer and tile drainage underlying these soil plots, leaching was minimal. Under normal field conditions in which leaching would be a more important factor, the downward movement of endrin might be greater. Another, less obvious fact is that the quantity of endrin in the surface 7.6 cm was less than the mean quantity between the 7.6- and 23-cm depths. This indicates that volatility and photodecomposition may have played a significant role in the dissipation of the endrin from the surface layers. The total amount of endrin remaining in the soil in 1966 was 28% of that remaining in 1954 following the last foliar application. When endrin was incorporated into and thoroughly mixed with the soil (Nash and Woolson, 1967), the total amount remaining in the soil in 1966 was 44% of that remaining 1954, three years following treatment. Thus, endrin persistence is greater when incorporated into the soil than when surface-applied to the crops. These figures also suggest that photodecomposition and volatilization are quite significant.

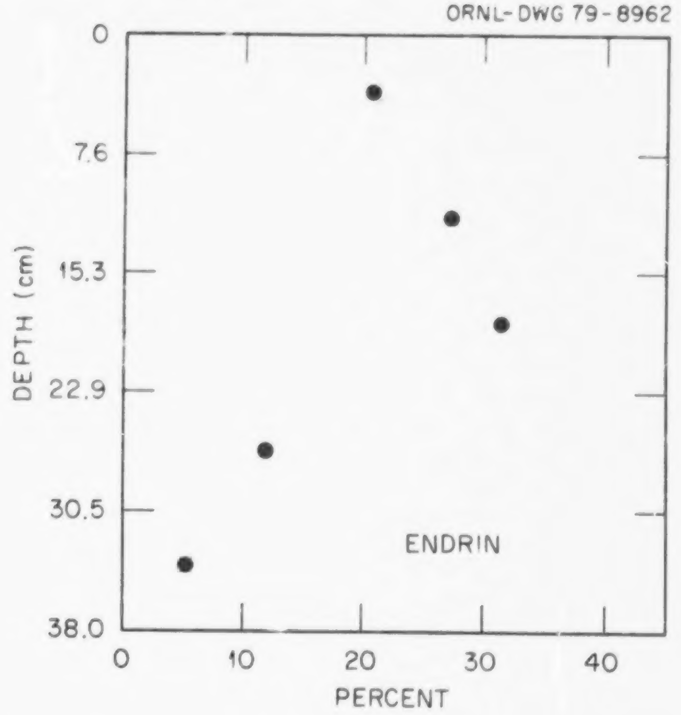

Fig. 7.11. Distribution of endrin in a 38-cm Congaree sandy-loam soil profile 13 years after the last foliar application. Source: R. G. Nash and E. A. Woolson, *Soil Sci. Soc. Am Proc.* 32: 525-527 (1968). Copyright 1968 by the Soil Science Society of America.

The disappearance of endrin from a soil surface in the field, as indicated by loss of biological activity, was investigated by Mulla (1960). Samples of Coachella fine sand were placed in wooden boxes and treated (to a depth of 6 in.) with endrin emulsion at a rate equivalent to 4.8 lb (2.2 kg) of toxicant per acre. After treatment, the boxes were placed in the ground, their tops flush with the surface, in an open vineyard in Southern California which was exposed to sunlight throughout the day. Soil temperatures ranged from lows of 38°F (3.3°C) at 6 AM and 10 PM to highs of greater than 120°F (49°C) at noon. The disappearance of endrin from the soil was determined by the mortality of *Hippelates colluson* (eye gnat) and *Culex quinquefasciatus* larvae (mosquito larvae) following exposure to the treated soil at intervals following endrin application. The data are presented in Table 7.44 and summarized in Fig. 7.12. The loss of endrin appears to follow the same trend in the tests conducted using both insects. After 48 hr the percentages remaining as indicated by eye gnat and mosquito larvae were 30 and 35 respectively. After 144 hr (six days) the figures were 25 and 22%. Half of the applied dosage was lost 24 hr after application. Since the toxicity of endrin degradation products for these species was not indicated and since degradation products were not measured, the possibility exists that some undetected degradation products also remained in the soil. Nevertheless, endrin did disappear

Table 7.44. Loss of endrin activity from the surface of soil in the field as tested against *Hippelates colluson* and *Culex quinquefasciatus* larvae

Treatment	*Hippelates colluson* Hours after treatment	Average No. gnats/ jar[a]	Reduction (%)	Toxicant recovered/ acre (lb)	Loss (%)
Endrin[b]	1	7	93	4.8	0
	24	53	49	2.1	56
	48	84	20	1.4	70
	96	84	20	1.4	70
	144	90	14	1.2	75
Control	1	104			

Treatment	*Culex quinquefasciatus* Hours after treatment	Average mortality of larvae (%)	Insecticide present[c] (ppm)	Average loss[c] (%)
Endrin[d]	1	100	0.0490	0
	24	81	0.0260	47
	48	50	0.0170	65
	96	16	0.0102	79
	144	19	0.0108	78

[a] Five replicates.

[b] Minimum quantity detectable is 0.7 lb/acre.

[c] Average of 3 tests each with 3 replicates per test. The percent loss was calculated from standard dosage mortality line for each material.

[d] Minimum quantity detected was 0.007 ppm.

Source: C. H. Brett and T. G. Bowery, *J. Econ. Entomol.* 51(6): 818-821 (1958). Copyright 1958 Entomological Society of America.

quite rapidly, probably as a result of volatilization from the sandy soil (low organic content) in the hot sun.

In conclusion, it appears that knowledge of the persistence of endrin in a particular soil at a given location may only be obtained with

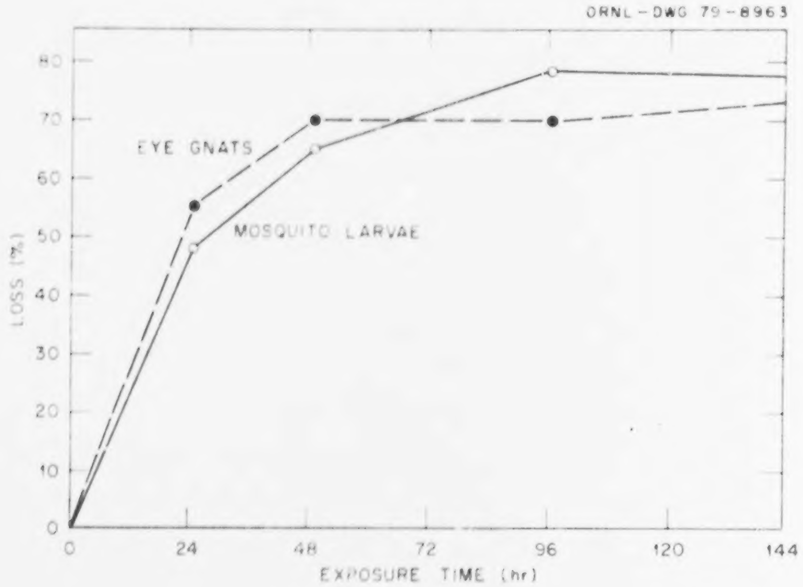

Fig. 7.12. Loss of endrin from surface of the soil as determined by bioassay procedure against eye gnats and mosquito larvae. Source: M. S. Mulla, *J. Econ. Entomol.* 53(4): 650–653 (1960). Copyright 1960 Soil Science Society of America.

certainty through investigations conducted with that soil at that location. Auxiliary studies may, however, provide guidelines as to what might be expected. Included among these guidelines are the following:

1. Endrin appears to be less persistent if applied to crops or to soil surface than if mixed throughout the soil.

2. Volatilization and photodecomposition are primarily responsible for the disappearance of endrin from soil surfaces.

3. Microbial degradation of endrin occurs anaerobically, and conditions which lead to a decrease in the soil oxidation-reduction potentional (such as flooding) accelerate endrin decomposition by this route. Microbial degradation is more significant at lower soil depths.

4. Cultivating the soil aids in the dissipation of endrin. Crop rotation is more effective than continuous cropping.

5. Soil organic content encourages persistence only when the percentage of organic matter is very large, as in the case of the muck soils used fot growing vegetables. Volatilization is most substantial from sandy soils.

6. Unless conditions are specified, it is not correct to label endrin as persistent in the lithosphere. Half-lives ranging from 24 hr to 11.8 years have been observed.

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8. ENVIRONMENTAL INTERACTIONS AND THEIR CONSEQUENCES

8.1 SUMMARY

Endrin enters the terrestrial environment primarily by deliberate application to crops. Associated routes of entry such as disposal and cleaning of equipment, vaporization, and surface runoff serve to transfer endrin to the aquatic and atmospheric compartments.

In the latest available figures (for the fiscal year 1973), the average daily intake of endrin by a man was 0.033 μg. This is at least two orders of magnitude below the WHO/FAO acceptable daily intake of 138.2 μg. Daily intakes of 1 μg or less have been reported since 1965, while the frequency of endrin occurrence in food composites has decreased from 2.8% in 1965 to 0.3% in 1973.

The concentrations of endrin found in water and air are also far below accepted limits, except for occasional high levels in the immediate vicinity of endrin usage. The maximum air concentration of endrin in 1975 was found in Jackson, Mississippi, 0.5 ng/m^3. The threshold limit value is 100 μg/m^3. And drinking water from a high endrin usage area in Louisiana contained a maximum endrin concentration of 23 ppt, less than one-fourth the suggested reasonable level of 100 ppt for potable water.

Processing of foods by storage, heating, freezing, and peeling root vegetables serves to lower endrin residues by as much as 80%. However, endrin residues in or on tobacco are not lost after curing (when approximately 40% of the residual endrin disappears); an average of 0.2 μg of endrin is present in a commercial cigarette.

Endrin has been shown to alter the nutritional value of some crops. Calcium, iron, and amino acid composition in wheat were reported to be significantly changed by soil concentrations of endrin as low as 10 ppm.

Biomagnification of endrin does not occur in succeedingly higher trophic levels of food chains. Bioconcentration does take place up to thousands of times the ambient concentrations. However, residue concentrations are based on partition between tissues, body fluids, and ambient concentration and are correlated to exposure levels and duration, solubilization in fat, and physiological changes.

Endrin resistance develops in a number of animals in response to chronic endrin exposure. The resistance apparently is controlled genetically and affords the resistant animal not only cross-resistance to many other organochlorine pesticides but also tolerance to higher endrin residues. It is these high residues (as much as 200 times the levels tolerated by susceptible animals) that pose a potential hazard to the environment. Both ingestion of prey with high endrin residues and release of the residues to ambient water cause mortality to other components of the ecosystem. Sublethal endrin effects such as reproductive, growth, and behavioral changes also disrupt the structure of a community.

Plants do not bioconcentrate endrin much above soil concentrations, but they translocate endrin from aerial parts to roots and soil as well as from soil to leaves and air. Thus, plants act to distribute endrin in the environment and expose more members of the ecosystem. The large biomass of plants makes them important in endrin cycling.

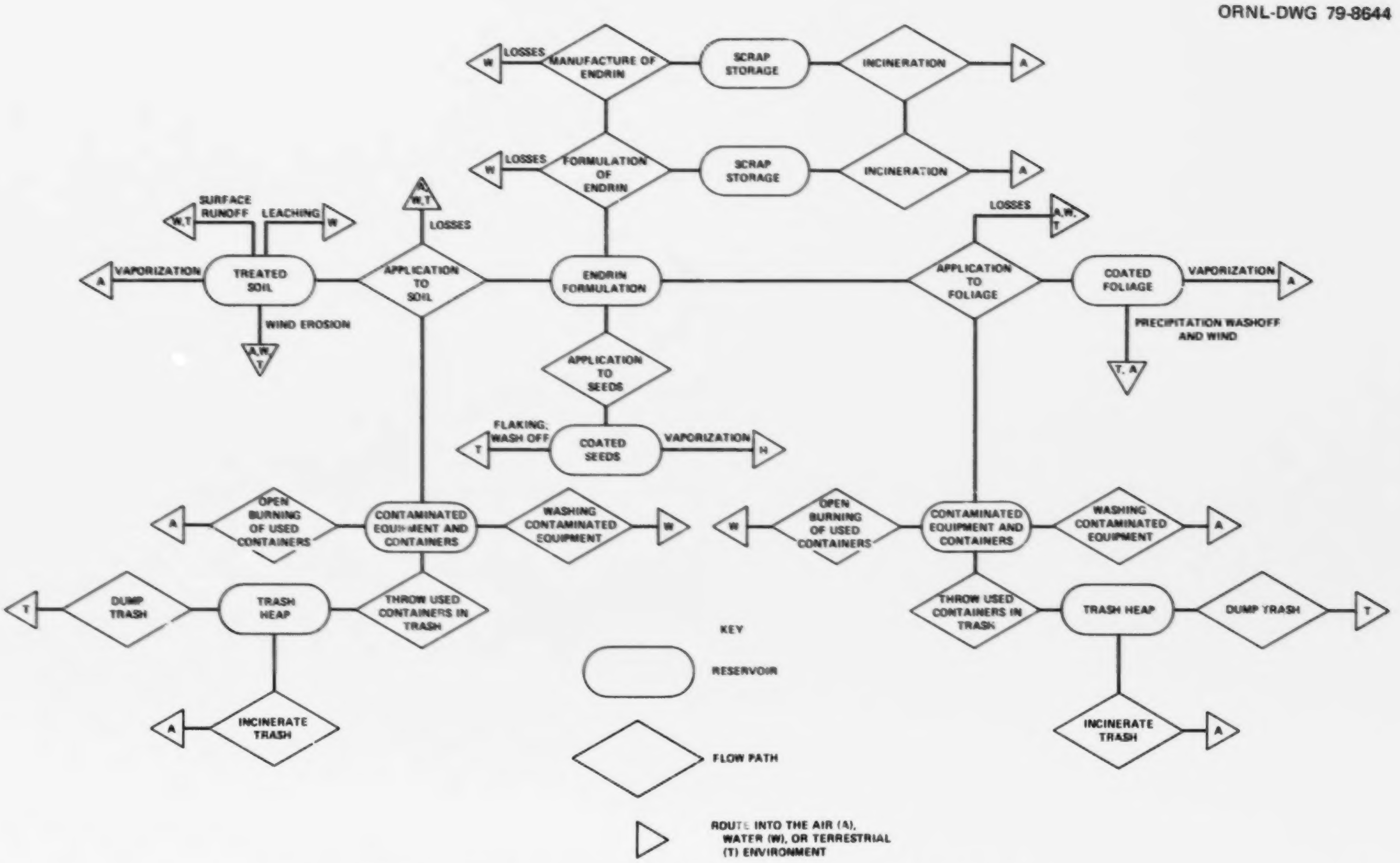
Worldwide monitoring data show that endrin occurs at very low levels, if at all. Since the massive fish kills in the Mississippi River and delta in 1963-64, the occurrence of endrin in the United States has steadily decreased. However, endrin has the highest acute toxicity of the common organochlorine insecticides, making even low levels of endrin a potential threat to the environment.

8.2 ENVIRONMENTAL CYCLING OF ENDRIN

Pesticides do not remain localized at the point of application but undergo cycling, by numerous pathways, throughout the envrionment. The function of a transport model is to define these pathways and estimate their relative importance in terms of frequency and quantity. Although the processes resulting in release of endrin to the environment and the mechanisms by which it is transported are known, the data necessary for a quantitative model are not available. Qualitative judgments as to which transport mechanisms predominate are, however, possible from analysis of both experimental and circumstantial evidence.

The routes by which endrin enters the environment are presented in Fig. 8.1. Although any one of these pathways might predominate under specialized conditions, vaporization from treated crops and soil, surface runoff, and direct application represent the major routes to the atmosphere, freshwater hydrosphere, and lithosphere respectively (Sects. 7.2 through 7.5).

An often overlooked, but unquestionably significant, route of endrin dispersion is the disposal and cleaning of contaminated containers and applicators. In one report, disposal of empty containers was listed as the third major source of endrin into the environment (U.S. EPA, 1972). The results of a survey to determine the disposal methods most frequently used by farmers are presented in Table 8.1. The most common procedure, open burning, undoubtedly results in volatilization of significant quantities of endrin, while the second most common procedure, rinsing and saving, is often carried out in streams, resulting in contamination of the hydrosphere (Saunders, 1969). Rinsing of spray equipment is usually performed at the same time. The third ranking practice, intermixing with other solid waste for dumping or incineration, leads to possible contamination of the lithosphere and the atmosphere respectively.


ORNL-DWG 79-8644
W
LOSSES
MANUFACTURE OF ENDRIN
SCRAP STORAGE
INCINERATION
A
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LOSSES
FORMULATION OF ENDRIN
SCRAP STORAGE
INCINERATION
A
W,T
SURFACE RUNOFF
LEACHING
W
A, W,T
LOSSES
A
VAPORIZATION
TREATED SOIL
WIND EROSION
A,W, T
APPLICATION TO SOIL
ENDRIN FORMULATION
APPLICATION TO FOLIAGE
LOSSES
A,W, T
COATED FOLIAGE
VAPORIZATION
A
PRECIPITATION WASHOFF AND WIND
T, A
APPLICATION TO SEEDS
T
FLAKING; WASH OFF
COATED SEEDS
VAPORIZATION
H
A
OPEN BURNING OF USED CONTAINERS
CONTAMINATED EQUIPMENT AND CONTAINERS
WASHING CONTAMINATED EQUIPMENT
W
W
OPEN BURNING OF USED CONTAINERS
CONTAMINATED EQUIPMENT AND CONTAINERS
WASHING CONTAMINATED EQUIPMENT
A
T
DUMP TRASH
TRASH HEAP
THROW USED CONTAINERS IN TRASH
THROW USED CONTAINERS IN TRASH
TRASH HEAP
DUMP TRASH
T
A
INCINERATE TRASH
INCINERATE TRASH
A
KEY
RESERVOIR
FLOW PATH
ROUTE INTO THE AIR (A), WATER (W), OR TERRESTRIAL (T) ENVIRONMENT

Fig. 8.1. Possible routes of endrin into the environment.

Table 8.1. Disposal methods used by farmers

Rank	Disposal method	Comment
1	Burn	Containers burned with other trash in open fire
2	Wash and save	Containers rinsed out and stored or reused
3	Throw in trash	Containers thrown for pickup with other solid waste
4	Bury	Farmer buries containers on his property
5	Discard	Farmers throw containers in ditch or field edge

Source: U.S. EPA, 1972.

A schematic representation of the movement of endrin through the environment following its release to the atmosphere, hydrosphere, or lithosphere is presented in Fig. 8.2.

While pathways such as surface runoff, irrigation, and dredging may be of significant consequence in terms of transport from one compartment of the environment to another, the effects, although often quite serious, are for the most part local. The only mechanisms able to account for the worldwide distribution of appreciable quantities of any pesticide are those which occur via the atmosphere. A typical cycle of atmospheric transport is presented in Fig. 8.3.

On one day in January 1965, 5 to 6 tons of dust per square mile was deposited in Cincinnati, Ohio, during a light rain. The dust originated in western Texas, where it had been picked up in a large wind stream on the previous day. At the time the dust fall occurred in Cincinnati, the dust cloud resulting from the storm stretched for 1500 miles in a 200-mile-wide band extending from the Gulf of Mexico to Lake Erie. The dust contained a total of 1.3 ppm chlorinated hydrocarbon insecticides (Frost, 1969).

The majority of endrin released or transported to the hydrosphere becomes adsorbed to suspended particulate matter and is eventually carried to the sediment. High levels of endrin residues in fresh water are almost always temporary, being associated with eroded soil particles which subsequently settle out (Frost, 1969). Although some redissolution may occur under normal circumstances, it is usually during periods of turbulence (such as heavy storms or dredging operations) that the sediment is sufficiently disturbed to effect resuspension of particulate matters with maximum release of pesticide back to the water. During such periods it would appear that significant river transport of endrin to the sea might

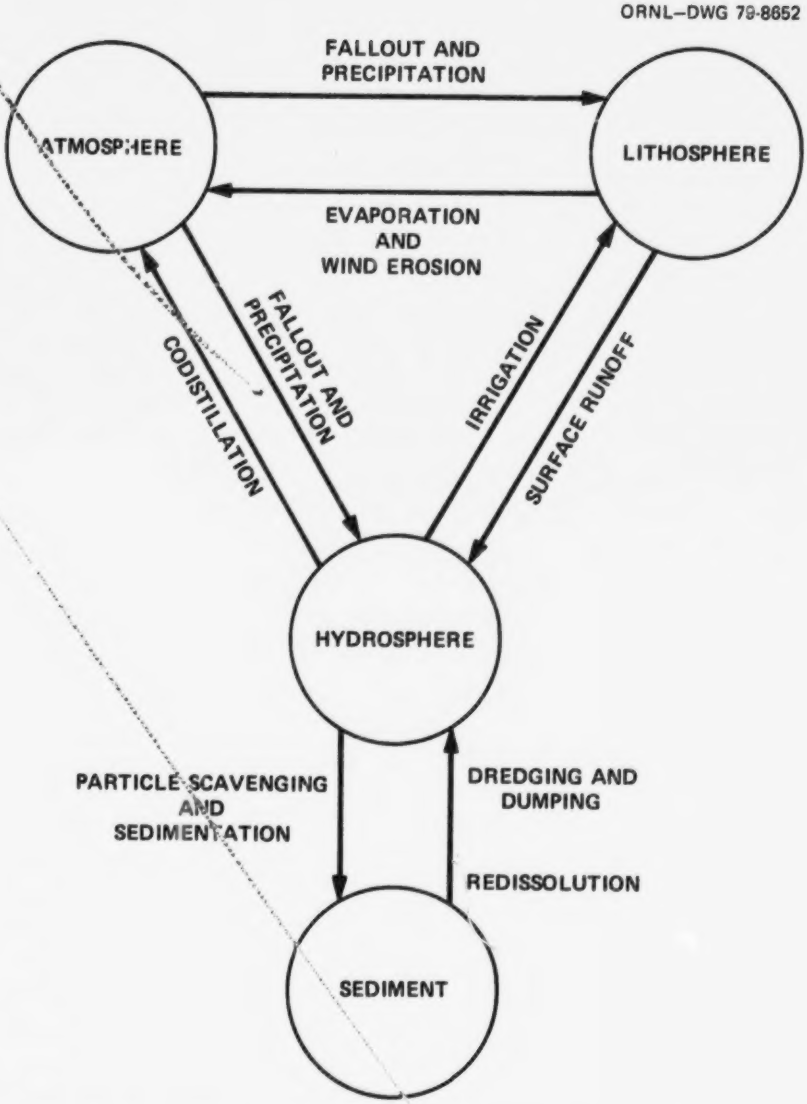
ORNL–DWG 79-8652
FALLOUT AND PRECIPITATION
ATMOSPHERE
LITHOSPHERE
EVAPORATION AND WIND EROSION
FALLOUT AND PRECIPITATION
CODISTILLATION
IRRIGATION
SURFACE RUNOFF
HYDROSPHERE
PARTICLE SCAVENGING AND SEDIMENTATION
DREDGING AND DUMPING
REDISSOLUTION
SEDIMENT

Fig. 8.2. Movement of endrin through the environment.

occur. However, resuspension and dissolution also carry the endrin up to the surface, where codistillation takes place. Furthermore, when the storm subsides, redeposition to the sediment follows. Thus, in actuality, only a small percent of the endrin released to freshwater bodies reaches the sea by this route.

Atmospheric transport provides the major route to the sea. While adsorption to the organic matter in the surface microlayer does occur with the reemission to the atmosphere (via codistillation and other processes) a distinct possibility, sedimentation once again predominates.

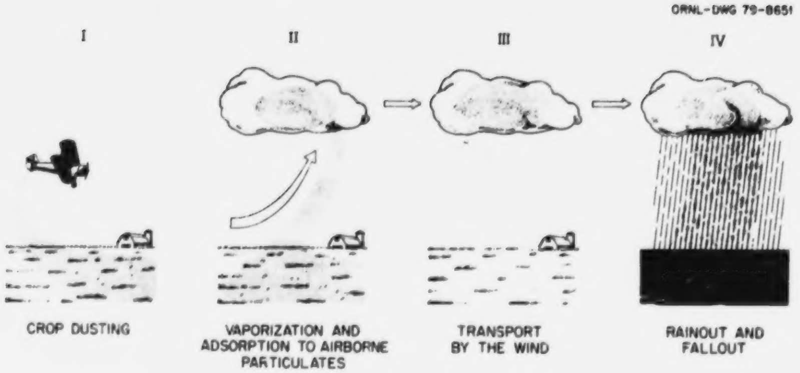

Fig. 8.3. Transport of vaporized endrin. Source: J. Frost, *Environment* 11(6): 14-29; 31-33 (1969). Copyright 1969 Wadham Publishers Ltd., Toronto, Ontario.

Due to the depth of the ocean, the chances for resuspension of contaminated sediment are, at best, limited. The sediment of the oceanic abyss, therefore, represents the ultimate sink for endrin and similar compounds. Whether these compounds remain dormant and relatively harmless, are degraded by microorganisms, or are ingested by bottom feeders is uncertain. The only real certainties are that the quantity of organochlorine insecticides and other toxic pollutants which are ultimately carried to the sea is enormous, and that the sea, while vast indeed, is not infinite.

8.2.1 Endrin in Food

Endrin residues may be ingested by man directly from plant material or from primary and secondary consumers of the contaminated plants. Man is also exposed to low levels of endrin in water and air. Potentially hazardous levels apparently occur only as a result of accidental mishaps such as spray drift or in the immediate vicinity of endrin use where high residues on plants or in water occasionally are detected.

The latest available figure for the average daily intake of endrin from food by a 69.1-kg man (June 1972 to July 1973) was 0.033 μg (0.5×10^{-6} mg/kg) (Bureau of Foods, 1975). This is well below the WHO/FAO acceptable daily intake of 138.2 μg (0.2×10^{-3} mg/kg) for a 69.1-kg man (WHO, 1973; U.S. EPA, 1974b).

While no standards have been set for water concentrations of endrin, 0.1 ppb is suggested as a reasonable limit for potable water (Schafer et al., 1969b). Occasionally, groundwater may contain more than 0.1 ppb, but high levels were correlated with precipitation and runoff following endrin application. Drinking water from Franklin, Louisiana, a region of high endrin usage, was found to contain a maximum of 23 ppt, well below 0.1 ppb (Lauer et al., 1966).

Exposure to endrin in the air has decreased from a maximum finding in 1971 in Greely, Colorado, of 25.6 ng/m^3 (U.S. EPA, 1971a) to a maximum in 1975 in Jackson, Mississippi, of 0.5 ng/m^3 (Kutz et al., 1976). No monitoring data contained endrin levels even approaching the threshold limit value of 100 μg/m^3 set by the American Conference of Governmental Industrial Hygienists for 1971 (Yobs et al., 1972).

In a series of analyses of total diets, the average daily intake of endrin in FY 1973 remained at the trace levels found in FY 1965 to 1970, but the frequency of occurrence decreased in FY 1973 compared with the six-year average of FY 1965 to 1970, 0.3 and 2.05% respectively (Table 8.2) (Bureau of Foods, 1975). The total diets were determined from market basket samples in four regions of the U.S. northeast, southeast, central, and west. A "market basket" consisted of approximately 117 individual food items grouped into 12 composites (Table 8.3) required for the total 14-day diet of a 15- to 20-year-old American male (69.1 kg) in the region of collection. All foods were normally treated before analysis, that is, meats were cooked, etc. The calculated daily intake of endrin by food class from FY 1964 to FY 1973 is given in Table 8.4. Only potatoes contained detectable endrin residues in FY 1973. Recommendations for acceptable daily intake, tolerances, and practical residue limits in food by WHO are given in Table 8.5.

Table 8.2. Average incidence and daily intake of endrin

FY	Positive composites (%)	Daily intake (mg)
1965	2.8	T[a]
1966	2.0	T
1967	1.7	T
1968	1.1	0.001
1969	3.3	T
1970	1.4	T
1971	0.6	T
1972	1.6	T
1973	0.3	T

[a] T = trace, <0.001 mg.

Source: Bureau of Foods, 1975.

Commercial catfish from Arkansas and Mississippi were reported to contain average residues ranging from 0.01 to 0.41 ppm. Four percent of the samples exceeded the FDA action level for maximum permissible endrin concentration in the edible portion of fish, which is 0.3 ppm (Hawthorne et al., 1974; Corckett et al., 1975). Humans may also be exposed to endrin in cow's milk and in steer, lamb, and hog meat (Sect. 6.5.1).

Table 8.3. Relative composition of diet by food class

Food group	Av/day	% of total diet
Dairy	769	22.6
Meats	273	8.0
Grain and cereal	417	12.3
Potatoes	200	5.9
Leafy vegetables	63	1.9
Legume vegetables	72	2.1
Root vegetables	34	1.0
Garden fruits	89	2.6
Fruits	222	6.5
Oil and fats	51	1.5
Sugars and adjuncts	82	2.4
Beverages	1130	33.2
Total	3402	100.0

Source: Bureau of Foods, 1975.

Endrin disappearance from growing and harvested crops is so variable that "half-life" data for endrin persistance on food plants should be viewed with skepticism (Hill, 1971). The largest factor in concentration decrease in plants is probably the dilution effected by plant growth (Hill, 1971). The loss of endrin residue depends on the sum of many factors, including temperature, volatization, metabolism, and dislodgement by wind and rain (Sect. 5.3.1). Since generalizations cannot be made that endrin on a given crop will always "disappear" at the same rate (Hill, 1971), only residue analyses on harvested crops can ascertain the potential hazard to humans.

Processing of some foods before human consumption significantly changed endrin residues. Endrin increased in soybean oils (0.28 ppm) relative to whole-crop levels (0.07 ppm) by the extraction process (Hill, 1970; U.S.D.A., Plant Pest Control Division, 1968). Storage above 12 weeks decreased endrin residues in Irish and sweet potatoes by 20% (Solar et al., 1971; Solar, 1971). Heat processing and freezing further lowered potato residues 65 and 52% respectively. Studies on turnips (Wheeler et al., 1969) and carrots (Hermanson et al., 1970) identified 50 to 80% of the endrin in the peels (Table 4.1).

The nutritional value of food may be affected by endrin. Thakre and Saxena (1970) reported that 10 to 30 ppm endrin in the soil decreased calcium uptake in wheat by 30 to 40% and increased calcium uptake in maize by 2.5 to 3 times. Iron uptake was not affected in maize but was decreased 40% in wheat by 20 to 30 ppm endrin. The amino acid composition was significantly changed in maize by 10 ppm endrin (Thakre and Saxena, 1972). The amino acids arginine, histine, leucine, proline, and lysine were increased two- to fivefold by 20 ppm or more, while a slight increase was noted in glutamine, asparagine, phenylalanine, glycine, serine, and tyrosine. Trytophan and valine were decreased by 33 and 10% respectively.

Table 8.4. Calculated daily intake of endrin by food class (mg/day)

Fiscal year	Meat, fish, and poultry	Potatoes	Leafy vegetables	Root vegetables	Garden fruit	Oils, fats, and shortening
1965		T[a]	T			
1966		T		T	T	T
1967		T	T	T		
1968		T				0.001
1969	T	T	T		T	
1970		T		T	T	
1971		T				
1972		T				
1973		T				

[a] T = trace, <0.001.

Sources: Corneliussen, 1969, 1970, 1972.
Duggan and Lipscomb, 1969.
Duggan et al., 1967, 1971.
Manske and Corneliussen, 1974.
Bureau of Foods, 1975.
Duggan and Weatherwax, 1967.

Table 8.5. Recommendations for endrin concerning acceptable daily intakes, tolerances, and practical residue limits as of November 1972

Maximum acceptable daily intake (mg/kg body weight)	Commodity	Residue limits (ppm)	
		Tolerances	Practical limits
0.002	Cottonseed, cottonseed oil (crude)	0.1	
	Corn (sweet)	0.02	
	Apples, barley, rice (husked and/or polished), sorghum, wheat	0.02	
	Edible cottonseed oil	0.02	
	Milk and milk products (fat basis)		0.02
	Fat of poultry		1.0
	Eggs (shell free)		0.2

Source: World Health Organization, *WHO Technical Report Series No. 525*, Geneva, 1973. Copyright 1973 World Health Organization.

Tobacco retains an average of 0.2 μg of endrin per commercial cigarette (Bowery et al., 1959). Forty percent of the residual endrin disappears during curing, but the remainder persists during cigarette manufacture. Endrin residues in chewing tobacco and pipe tobacco increased approximately threefold from 1969 (0.05 ppm) to 1971 (0.114 ppm) (Domanski et al., 1973). Cigar and snuff residues remained at approximately 0.06 and 0.16 ppm, respectively, from 1969 to 1972 (Domanski et al., 1973; Domanski and Guthrie, 1974). Cigarette residues decreased from 0.18 to 0.09 ppm from 1969 to 1971 (Domanski et al., 1973).

Respiratory and dermal exposure of a dust or spray machine operator, estimated by Wolfe et al. (1963), is shown in Table 8.6. In all operations studied, calculations indicated that the dermal exposure was greater than the respiratory exposure. Potential dermal exposure during orchard spraying with 0.05% spray (1.2 lb active ingredient/acre) ranges from 2.5 to 3 mg/hr and for respiratory exposure reaches 0.01 mg/hr (Wolfe et al., 1963; Wolfe et al., 1967). Higher levels of exposure occurred during dusting of potatoes with 1% endrin dust, where levels of 18.5 mg/hr for dermal exposure and 0.41 mg/hr for respiratory exposure were found (Wolfe et al., 1963). During the spraying of row crops, a dermal exposure of 0.15 mg/hr was found, but the respiratory exposure was below the limits of detection of the analytical method employed (Jegier, 1964). The EPA worker protection standards for agricultural use of endrin are given in Table 8.7.

Table 8.6. Exposure of workers to endrin

Activity	Dermal (mg/hr)	Respiratory (mg/hr)	Total % toxic dose (hr)
Spraying orchard cover crops for mouse control	2.6	0.01	0.21
High-pressure power handgun	3	0.01	0.25
Operating power air blast or boom sprayers	2.5	0.01	0.21
Dusting potatoes	18.7	0.41	1.5
Spraying row crops	0.15		
Piloting airplane during air application	1.18	0.08	0.29

Source: H. R. Wolfe, W. F. Durham, and J. F. Armstrong, *Arch. Environ. Health* 14(4): 622-633 (1967). Copyright American Medical Association.

Table 8.7. U.S. Environmental Protection Agency worker protection standards for agricultural use of endrin

General standard:

No endrin should reenter a field treated with endrin for at least 48 hr

Tolerances for endrin residues:

A tolerance of zero for residues in or on the following raw agricultural commodities:

- broccoli
- brussels sprouts
- cabbage
- cauliflower
- cottonseed
- cucumber
- eggplant
- peppers
- potatoes
- sugar beets, sugar-beet tops
- summer squash
- tomatoes

Source: U.S. EPA, 1971c.

8.2.2 Terrestrial Ecosystems

8.2.2.1 Introduction. In tracing the movement of endrin in terrestrial food chains, both direct and indirect reactions between trophic levels must be anticipated. A well-planned food web may easily fall apart under the biocidal and physiological effects exerted by endrin (Fig. 8.2). Sources, transport in and out, and persistence of endrin in a terrestrial ecosystem have been discussed in Sect. 7.5.

Factors influencing the introduction of endrin into the environment may be artifical or natural. Certainly, economics is the chief artificial factor. The return on a one-dollar expenditure for agricultural pesticides was approximately four dollars according to J. C. Headly (Decker, 1974). But not only profit is involved. Crop demands continually increase, and more produce must come from the same or shrinking land area. Thus, pesticide residues of some type are likely to remain, and it becomes a matter of wisely choosing the most beneficial and least hazardous compound. Other artificial factors include the type of endrin formulation (granules, dust, and emulsion) with its associated persistence and toxicity. As would be expected, formulations with a longer active life (granules) exert less toxicity than formulations with shorter effective duration (emulsions), even though the endrin concentration is equal (Iyatomi et al., 1958).

Natural factors influence endrin residues in a subtle way. They include soil type, absorption by plants or animals, weather, and food-chain transfers, among others. The longer a compound remains in the environment, the greater is the likelihood of movement out of the target area in soil, air, water, and migrating carriers (Fig. 8.4). Prolonged exposure also allows development of resistence and natural selection processes in the biota.

Currently, scientists are more competent to confirm the presence of endrin than to assess its ecological impact. The consensus appears to be that man and most other mammals are able to cope effectively with ingested endrin, but that many birds and cold-blooded animals are less able to do so (Decker, 1974). Food-chain studies tend to show that predators at the highest trophic levels are in the most danger. However, the knowledge of endrin's actions in terrestrial food chains is scant and incomplete, lacking the data to elucidate potentially hazardous trends. While there is no doubt of endrin's toxicity to most fauna, it usually is true that species extinction is only likely to occur if the population at risk is already in a perilous situation for other reasons (Edwards, 1973).

Pesticides often set off an unpredicted sequence of events due to toxic effects on nontarget organisms. The response of nontarget species usually has not been evaluated and poses the greatest hazard in pesticide use. Thus, information in addition to lethal doses is needed to determine populational, trophodynamic consequences (Finlayson and MacCarthy, 1973).

Endrin influences the food-chain transfers by both immediate, differential mortality and by long-term accumulation of residues (Khan

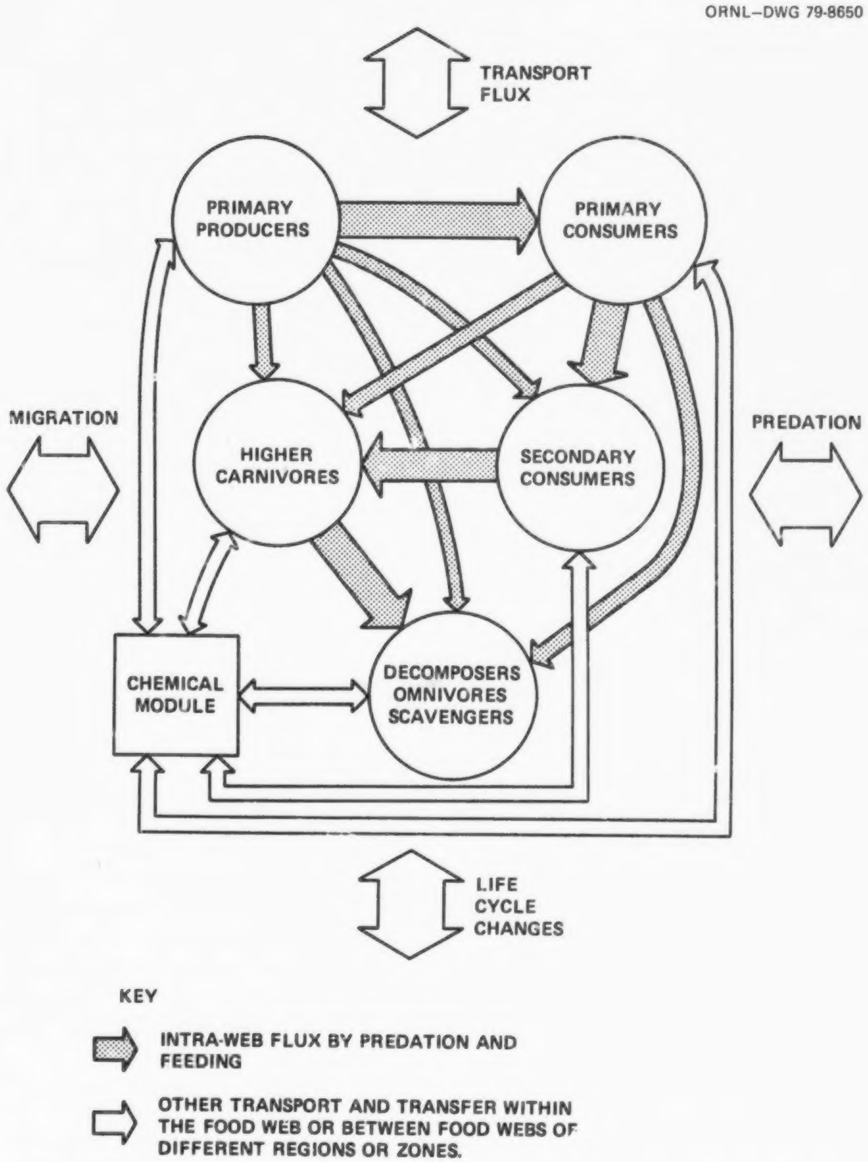
ORNL–DWG 79-8650
TRANSPORT FLUX
PRIMARY PRODUCERS
PRIMARY CONSUMERS
MIGRATION
HIGHER CARNIVORES
SECONDARY CONSUMERS
PREDATION
CHEMICAL MODULE
DECOMPOSERS OMNIVORES SCAVENGERS
LIFE CYCLE CHANGES
KEY
INTRA-WEB FLUX BY PREDATION AND FEEDING
OTHER TRANSPORT AND TRANSFER WITHIN THE FOOD WEB OR BETWEEN FOOD WEBS OF DIFFERENT REGIONS OR ZONES.

Fig. 8.4. Food web module. Source: Gillett et al., 1974.

et al., 1971). Some of the impacts of endrin on the terrestrial ecosystem include (Pimental and Goodman, 1974):

- influence on structure and function of a community by differential toxicity,
- reduction of population numbers,
- alteration of natural habitats by effects on flora and fauna,

- changes in normal behavior patterns,
- stimulation or suppression of growth,
- increase or decrease in reproduction,
- alteration in nutritional content and preference of food,
- increase or decrease in susceptibility to disease and predation,
- change in the natural evolution of a species population.

In the terrestrial ecosystem, the major pathway of endrin uptake is feeding on treated vegetation or contaminated prey. The apparent lack of endrin storage in plants and animals makes biomagnification of endrin by food-chain transfers less aggravated. But short-term endrin buildups present a source of food with high endrin residues that can cause predator mortality. This is particularly true of endrin in small animals (Craig and Rudd, 1974). Small predators take in higher endrin residues per body weight than do large predators because small prey have increased surface area for absorption of endrin. In addition, the higher metabolic rates in small animals require greater food intake (and endrin residues) per gram of body weight than do larger animals, creating a hazard both to self and predator (Craig and Rudd, 1974).

8.2.2.2 Plants. Transport of endrin in the terrestrial food chain, using plants as an intermediary vehicle, is important because of the large biomass of plants. Endrin residues in or on plants may originate from accidental or intentional contamination. Accidental sources include volatilization from soil and drift from aerial application. However, most plant residues result from deliberate treatments used to control insects.

Plants accumulate endrin from leaf and soil applications, but not to concentrations much in excess of ambient levels (Sect. 4.3). Uptake depends upon plant species, soil type, and soil concentrations of endrin (Table 4.2). Endrin applied to plant leaves or soil was translocated throughout the plant, occurring in ever decreasing concentrations at distances increasingly further from the source. Endrin was found in the soil after application to aerial parts of plants, demonstrating release as well as uptake of endrin by plants. Thus, plants make endrin available to both herbivores and soil organisms.

Endrin treatment (4 and 2 oz/acre, 0.28 and 0.14 kg/ha) of an alfalfa-bromegrass field produced the highest residues (0.87 and 0.55 ppm respectively) when applied 14 days before harvest (Gyrisco and Huddleston, 1961). Treatments at 21 and 10 days before harvest reduced residues approximately 40 and 50% respectively. Alfalfa treated immediately before harvest had endrin residues reduced 30-fold by air-drying three days, plus one day of oven-drying. Thus, timing of endrin application and processing before use as fodder can substantially reduce the amount of endrin ingested by livestock. However, Archer (1968b) determined that endrin residues were located in the cuticle of alfalfa. Heat removed 35% of the residue, and adding water doubled the loss.

8.2.2.3 Invertebrates. Endrin was used as an effective poison for snails (*Achatine fulica*) that attacked crops in India (Manna and Ghose, 1972). Baits were prepared by combining different amounts of endrin with wheat bran and molasses. Snail mortality was directly related to the percent of endrin in the bait; seven-day periods of feeding 2.0, 1.0, 0.5, and 0.25% endrin produced 100, 90, 40, and 10% mortality respectively. Endrin damaged the snail gut, inhibited mucus secretion, and interfered with peristalsis. No residue determinations were made by Manna and Ghose (1972), buth Thompson (1973) reported endrin residues of 0.90 ppm (dry weight) in snails from soils containing 1.74 ppm. Gish (1970) determined endrin residues of 2.72 ppm in snails from locations of 3.47 ppm in the soil. The data available suggest that snails do not bioaccumulate large amounts of endrin relative to the soil concentration.

Bioaccumulation factors for endrin in slugs were considerably higher than those for snails (Gish, 1970; Thompson, 1973). Slugs collected in endrin-treated orchards contained residues as high as 134 ppm when soil residues were 3.47 ppm (Gish, 1970). Generally, lower soil residues (0.013 ppm) produced lower residues in slugs (1.14 ppm), but bioconcentration factors increased (88-fold in 0.013-ppm soil compared with 39-fold in 3.47-ppm soil).

There was no significant relationship between endrin residues in soil and earthworms (Gish, 1970). As an illustration, earthworms collected from cotton fields contained endrin residues of 0.20, 0.23, 5.40, 9.40, and 11.04 ppm in soils of 0.024, 1.29, 0.11, 0.09, and 3.47 ppm respectively. Bioconcentration factors ranged from 14.0 to 103.3 (Gish, 1970). Soil invertebrates, and especially slugs and earthworms, provide a first step in endrin transfer to birds, moles, shrews, and snakes. Additionally, fecal matter and decomposition spread endrin in the soil.

Information on endrin residues in insects was not available, but endrin is absorbed through body surfaces and by ingestion (Sect. 5.5). Undoubtedly, surface residues account for the major source of endrin transfer to other trophic levels in the food chain by insects. Cotton leafworms exposed to endrin concentrations of 19.5% developed a degree of tolerance correlated to the duration of exposure (Hassan et al., 1970).

The economic benefits of endrin in pest control are particularly important. Some of the crops treated with endrin are: cabbage (Harris and Svec, 1970; Gupta and Sharma, 1971), soybeans (Lee, 1962), eggplant (Pal, 1971), cotton (Khan et al., 1971; Misra, 1970; Loutfy et al., 1970), rice (Israel et al., 1969), wheat (Bindra and Singh, 1973; Khan et al., 1972), sugarcane (Sithanantham and Daniel, 1972; Kavadia et al, 1970), and corn (Bindra and Singh, 1973). Residue levels in food plants are discussed in Sect. 8.2.1.

8.2.2.4 Vertebrates. Almost all mammals appear to have the ability to accumulate endrin. Data on endrin residues in wild animals are essentially from isolated samples, except for birds. This probably reflects the low endrin levels encountered away from immediate application areas, as well as the obvious problems involved in collecting large sample numbers.

Continuous feeding of 2.0 ppm endrin to steers, lambs, and hogs produced accumulations of 1.0 ppm in fat tissue (Brooks, 1969). Forty Wisconsin dairy herds had endrin residues in milk of 0.25 ppm (fat basis) in 1964 to 1967 (Moubry et al., 1968a). Endrin residues in cow milk were directly related to the duration and level of exposure to endrin-treated hay, with a minimum daily endrin intake of 20 mg as forage residue before endrin detection in milk (Ely et al., 1957). Lambs grazing on pasture treated with endrin granules accumulated up to 23.7 ppm in external fat deposits without showing adverse effects (Long et al., 1961). In all cases, withdrawal of endrin was followed by a decrease in endrin accumulations.

Residues detected in wild birds cannot be related to specific intake concentrations, but they are indicative of the distribution of a compound. A report of pesticide residues in eagles (Reichel et al., 1969a) revealed a total of 6 of the 29 bald eagles analyzed had endrin residues in liver tissue with a median value of 0.09 ppm. Brain tissue contained a trace of endrin, while a level of 0.09 ppm was determined for the carcass. Endrin was detected in a composite sample of brain, heart, kidney, liver, and muscle tissue at a medial level of less than 0.1 ppm in four golden eagles (Reidinger and Crabtree, 1974). No endrin was detected in brain tissue alone, and of ten adipose samples containing endrin, the highest residue detected was 0.3 ppm with a median of less than 0.1 ppm for the other nine samples.

An analysis of brain tissue from common egrets found dead or moribound at the Audubon Canyon Ranch in California during 1970 revealed endrin residues in all five of the birds tested. A mean level of less than 0.18 ppm was reported with a range of less than 0.10 to 0.28 ppm detected. It was revealed that endrin was still being used at the time to treat conifer seeds as a protection against seed-eating mice in the forest regions, but whether this was the principal dietary source of the endrin detected was not clear (Faber et al., 1972).

Ring-necked pheasants from southwestern Idaho collected during 1969 were analyzed for endrin content of adipose tissue and eggs. Only trace amounts were detected in the samples (Messick, 1972). Juvenile pheasants from southeastern South Dakota were collected and tested during July 1967. The brain tissues of the birds as well as the plant and animal material from the crop contents revealed no endrin residues at a detection limit of 0.03 ppm (Linder and Dahlgren, 1970). No endrin was detected at a minimum limit of 0.05 ppm in adipose tissue from pheasants or sharp-tailed grouse from South Dakota (Greichus et al., 1968).

Songbirds from regions in California where endrin was used contained residues in muscle (1.23 ppm average) and gizzard (1.45 ppm average) (Keith and Hunt, 1966). Waterfowl from the same regions had only a trace (0.03 to 0.07 ppm) of endrin in fat and egg yolk. Sixty-eight percent of avian samples from the Gulf coast of Texas had endrin residues (Flickinger and King, 1972). The birds included waterfowl, shorebirds, and passerines. The highest (0.7 ppm) and most frequent (100%) residues were found in geese, and the least amount of endrin (0.1 ppm or less in 15%) was found in shorebirds (stilt, rail, and sandpiper, for example). Of the aquatic food sources tested, only one sample of crayfish contained traces of endrin. This suggests that local food sources were not responsible for the endrin accumulations, since endrin was not being used in field applications.

These experimental data indicate that the residues of endrin in wild birds appear to be quite low if detectable at all. However, Health et al. (1972) found endrin to be the most toxic of the 89 chemicals tested in bobwhites, Japanese quail, ring-necked pheasants, and mallards. If this toxic ranking of endrin is applicable to all avians, levels similar to those reported in food sources may be sufficient to induce injury or death.

8.2.3 Aquatic Ecosystems

8.2.3.1 Introduction. The introduction of endrin into aquatic ecosystems presents a hazard to the environment, ranging from the microorganisms, which are necessary for decompositon and cycling of nutrients, to man, who may eventually consume harmful amounts of endrin residues in fish and shellfish. The Environmental Defense Fund has indicated that concentrations in water in excess of 2 ppt (2 μg/liter) would result in consumption of quantities of endrin, by human populations relying heavily upon fish, in excess of the WHO/FAO acceptable daily intake of 2×10^{-4} mg/kg per day (U.S. EPA, 1974b).

Bioconcentration (the ability of an organism to concentrate a substance against a gradient) by aquatic organisms is the immediate cause of the adverse effects observed in aquatic food-chain relationships, while biomagnification (the increasing concentrations of a substance with increasingly higher trophic levels) per se is relatively unimportant. As presented in Sect. 5.2., the data support equilibrium reactions between ambient and organismal endrin levels as the basis for ultimate endrin concentrations. Endrin has the highest acute toxicity to aquatic organisms of the common organochlorine insecticides, causing mortality at concentrations less than 1 ppb. Bioconcentration factors of 1000 or more are commonly found in fish and aquatic invertebrates, resulting in the ingestion of high, and often fatal, endrin concentrations by predator species. Louisiana's brown pelican population disappeared entirely in the early 1960s, and three-fourths of a restocking effort was eliminated in 1975 as a result of lethal endrin doses presumably acquired from fish (Anonymous, 1975c).

In the process of controlling insect populations by endrin, natural insect predators are also eliminated. Field applications of endrin at 0.5 lb/acre (0.55 kg/ha) completely killed all bullfrog tadpoles (*Rana catesbeiana*) (Sanders, 1970). Frog (*Pseudacris triseriata*) and toad (*Bufo woodhousii*) tadpoles were more sensitive to endrin than to other common insecticides such as lindane, DDE, and dieldrin (Sanders, 1970).

A series of processes influence the bioconcentration of endrin in aquatic organisms. The concentration of endrin in the water is directly related to the bottom concentration, with sediment effectively removing endrin from the water and the adsorption capacity increased by the amount of organics present. Sediment has been described as "a way station, not a graveyard" by Hamelink (1971), because decreasing water concentration will release endrin from the sediment until an exchange equilibrium is again reached.

Algae also act as adsorbants of endrin, with their endrin concentrations dependent not only on water concentration but the amount of adsorbants present. For example, more endrin is adsorbed per algal unit weight when the standing crop is low than when it is high, assuming the endrin concentration remains constant (Hamelink, 1971).

Both invertebrates and fish act as multiphase exchange systems. Because invertebrates, such as oysters, accumulate endrin an order of magnitude higher than do algae, it is often assumed that the source is from the algal food. However, it appears that the endrin concentration in algae and invertebrates is unrelated, except for a mutual correlation with the water concentration (Hamelink, 1971). Fish bioaccumulate endrin from food and by absorption form water (Sect. 5.2.2). The food probably contains the major amount under natural conditions, since it may be bioconcentrated in invertebrates to levels far above that it water. However, the residue concentration in fish tissue eventually depends on the water concentration, since equilibrium is reached in the different exchange phases of tissue-blood-water. Residue equilibria, in turn, are affected by the amount of fat present for solubilization of the highly lipid-soluble endrin and the length of time available for exchange to take place. Thus, Hamelink (1971) and Johnson (1968) suggest that endrin accumulation is ultimately controlled by partitioning (i.e., solubility differences between water and lipid) in fish and invertebrates.

As endrin solubility is increased in waters, residue levels in organisms should decrease if, indeed, endrin bioaccumulation is controlled by solubility differences. Hamelink (1971) states that low concentration factors between water and fish are found in turbid, highly eutrophic water (water with high organic content), supporting the partition role in endrin biomagnification. Sediments in eutrophic waters are in a reductive state, due to the oxygen depletion. Thus, microorganisms probably contribute to the lowering of endrin levels in water and, therefore, tissues by reductive degradation of the compound.

Aquatic ecosystems present a number of conditions that tend to intensify the effects of endrin pollution. Among these are the following:

- maximized exposure because the medium containing endrin surrounds the organisms at all times,
- adsorption of endrin onto algal cells, resulting in concentration factors at least an order of magnitude higher than ambient levels,
- long food chains creating more steps in the short-term biomagnification residues,
- oceans and lakes acting as endrin sinks.

The total impact of endrin on aquatic ecosystems depends on its effect on each member of the food chain, whether directly, through predation, or by differential toxicity. The readily apparent effects of endrin may overshadow less obvious ones. For example, alterations in normal behavior (such as hyperactivity) may lead to exposure of the organism to predation; and changes in developmental timing may prevent emergence during favorable times, leading to eventual extinction (Jensen and Gaufin, 1966).

In general, endrin pollution patterns parallel drainage basins of intensive agricultural areas, and the seasonal distribution of endrin in aquatic environments supports an agricultural origin. Levels begin to rise in the late spring and peak during the summer (Butler, 1971). This appears to reflect endrin application and maximum runoff during the first precipitations following use. The seasonal maximum of endrin in aquatic ecosystems unhappily coincides with the time of maximal numbers of larval fish and shellfish (Butler, 1971). Thus the danger of a single large pollution incident wiping out an entire season of larvae is present. Of secondary importance in endrin pollution of aquatic environments is drainage from large urban areas and from industrial plants formulating endrin.

8.2.3.2 Plankton. Endrin may significantly influence succession and dominance of species within a phytoplankton community by not being equally toxic to all organisms. Marine phytoplankton isolated from markedly different oceanic environments showed varying responses to endrin (Menzel et al., 1970; Pfister, 1972). The isolates were *Skeletonema costatum* (located in the coastal diatom), *Dunaliella tertiolecta* (in estuarine green flagellate), *Coccolithus huxleyi* (in oceanic coccolithophorid), and *Cyclotella nana* (in oceanic diatom). *C. huxleyi*, *S. costatum*, and *C. nana* had cell division and carbon uptake reduced by endrin concentrations of 100, 10, and 1 ppb, respectively, while *D. tertiolecta* was not affected at concentrations up to 1000 ppb (Fig. 3.5). The differing responses may reflect the need for adaptability in the face of unstable environmental conditions. Fluctuating estuarine conditions acclimate the inhabitants to stress and the need to respond, as illustrated by the low sensitivity of *D. tertiolecta* when compared with oceanic species from relatively unchanging habitats.

Productivity (as measured by carbon incorporation) of a natural phytoplankton community was decreased 28 to 46% by exposure to 1.0 ppm endrin for 4 hr (Butler, 1963). This suggests that subtle changes may be occurring even while microorganisms are accumulating and apparently resisting endrin. Any interference with primary productivity will have drastic and far-reaching consequences in a trophic pyramid (Fig. 8.5).

8.2.3.3 Invertebrates. Aquatic invertebrates accumulate endrin mainly by absorption from water but also from sediments and food sources, such as plankton. After a rapid uptake, tissue concentrations decrease progressively until apparently an equilibrium is reached with ambient endrin. For example, *Culex pipiens quinquefasciatus* had decreasing bioconcentration factors of 2100, 500, and 310 on days 1, 2, and 3 respectively (Metcalf et al., 1973). The similar endrin residues in the associated components of two streams also suggest the attainment of

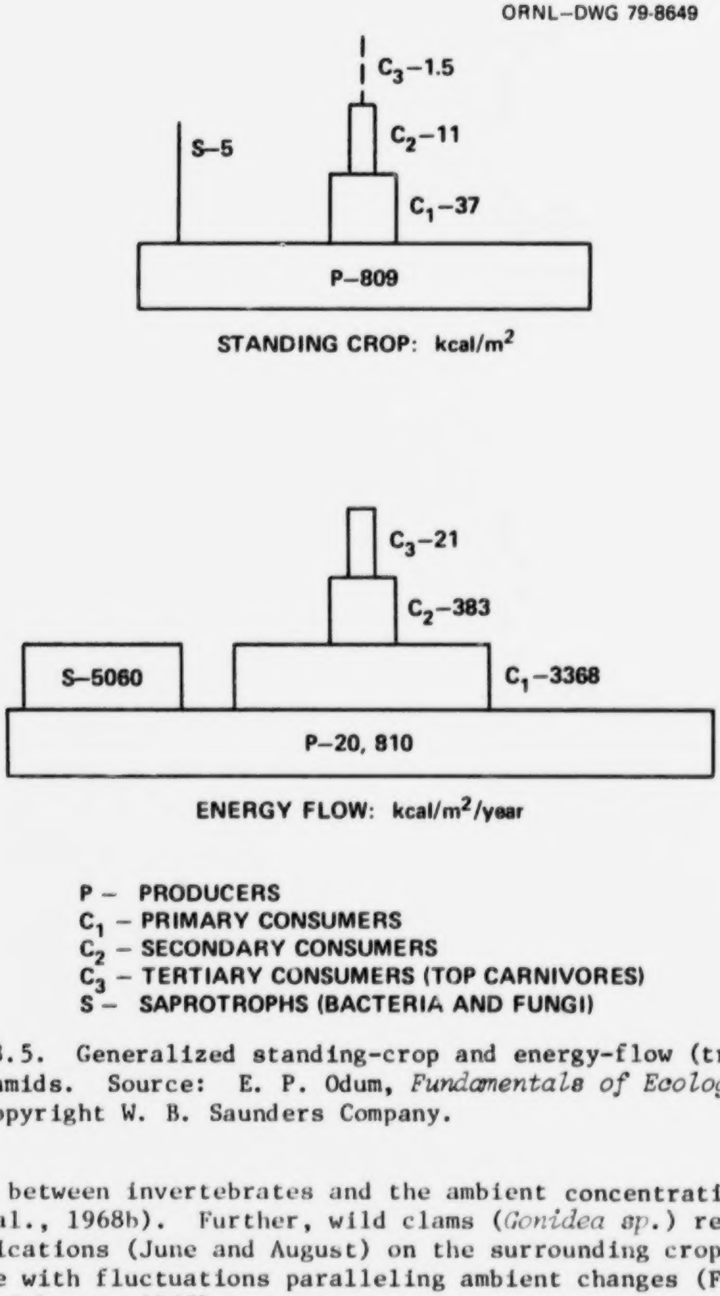
ORNL–DWG 79-8649
C_3–1.5
C_2–11
S–5
C_1–37
P–809
STANDING CROP: kcal/m^2
C_3–21
C_2–383
S–5060
C_1–3368
P–20, 810
ENERGY FLOW: kcal/m^2/year
P – PRODUCERS
C_1 – PRIMARY CONSUMERS
C_2 – SECONDARY CONSUMERS
C_3 – TERTIARY CONSUMERS (TOP CARNIVORES)
S – SAPROTROPHS (BACTERIA AND FUNGI)

Fig. 8.5. Generalized standing-crop and energy-flow (trophic level) pyramids. Source: E. P. Odum, *Fundamentals of Ecology*, 3rd ed. (1971). Copyright W. B. Saunders Company.

equilibrium between invertebrates and the ambient concentration (Table 8.8) (Moubry et al., 1968b). Further, wild clams (*Gonidea sp.*) responded to endrin applications (June and August) on the surrounding cropland by rapid uptake with fluctuations paralleling ambient changes (Fig. 8.6) (Godsil and Johnson, 1968).

Response to ambient endrin concentration was also demonstrated by rainfall and tissue residue correlation in freshwater mussels kept in an endrin-polluted pond (Ryan et al., 1972). The lowered endrin residues in the pond (after a heavy rainfall caused water to overflow the dam) were reflected in the decreased endrin concentration in mussel tissues (Fig. 8.7).

Table 8.8. Endrin residues from two anastomosing creeks in Wisconsin

Location/organism	Endrin residues (ppm dry weight)	
	Silt	Debris
North Branch	0.003	0.025
Alderfly larvae	0.009	
Caddisfly larvae	0.003	
Freshwater shrimp		0.025
South Branch	0.002	0.011
Composite of bottom organisms	0.004	
Confluence	0.013	0.014
Freshwater shrimp		0.013

Source: data from Moubry et al., 1968b.

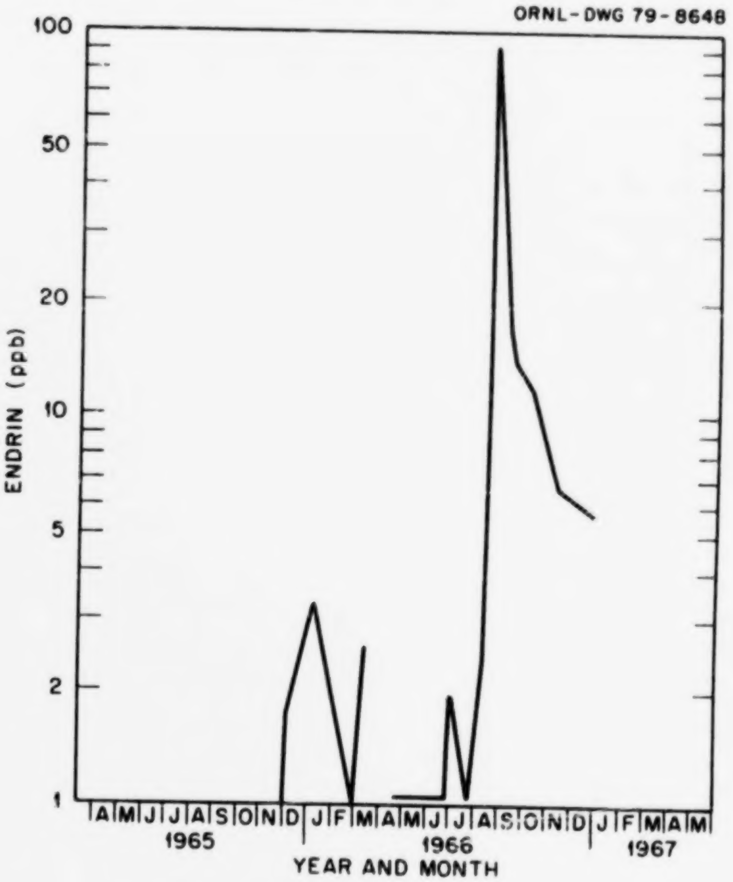

Fig. 8.6. Endrin accumulation in clams. Source: Godsil and Johnson, 1968.

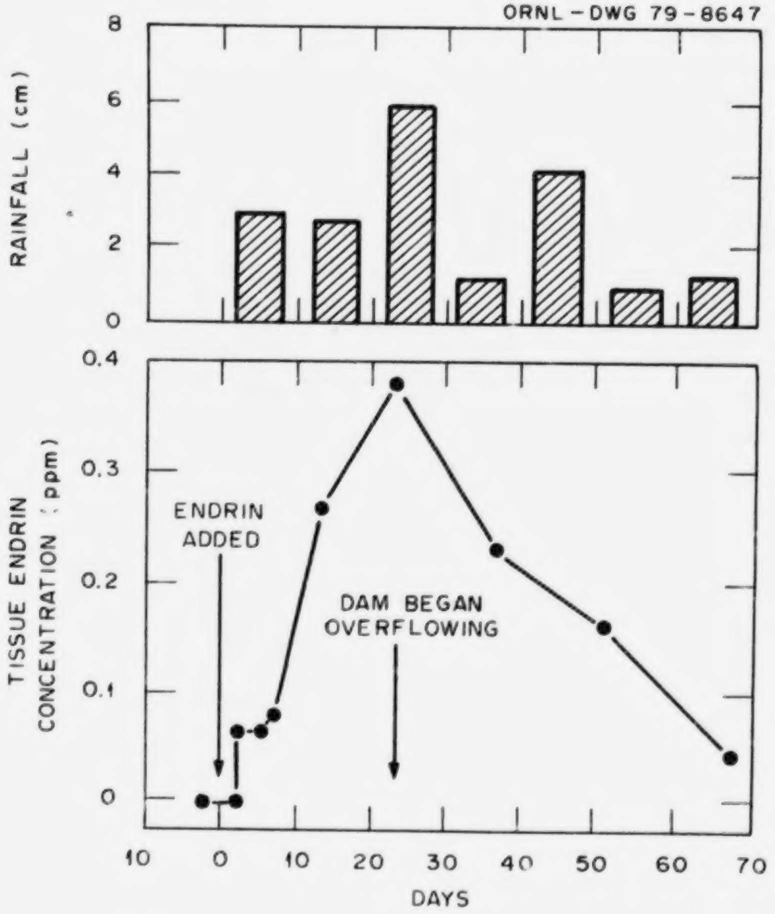

Fig. 8.7. Endrin concentration in mussel tissues and rainfall during ten-day periods of study. Source: S. Ryan, G. J. Bacher, and A. A. Maryin, *Search* 3(11/12): 446-447 (1972). Copyright 1972 Australian and New Zealand Association for the Advancement of Science.

Another factor involved in bioaccumulation of endrin by invertebrates appears to be the surface-area-to-volume ratio, suggesting that direct absorption contributes to body burdens. Under identical experimental conditions, higher endrin concentrations occurred in water fleas (with the larger area/volume ratio) than in mosquito larvae (Metcalf et al., 1973). However, species differences may complicate the relationship.

The rapid adjustment to ambient endrin concentrations makes mollusks a good monitoring organism for endrin pollution (Modin, 1969; Butler, 1971), but several factors must be considered before interpreting the results. The need to use consistent sample species is illustrated by the varying rates of uptake and elimination in different species. For example, substituting the hardshell clam (*M. mercenaria*) for the softshell clam (*M. arenaria*) results in approximately a one-third decrease in bioaccumulation and elimination rates (Duke and Dumas, 1974; Butler, 1971). Sequential data are necessary to draw valid conclusions from bioaccumulation levels. Heavy rainfall may flush out an estuarine system almost overnight and, concurrently, tissue residues. A series of samples would be needed to correlate tissue cleansing with rainfall and not assume a drop in pollution input. Other anomalous cases of bioaccumulation as a mirror of endrin pollution may result from cycling of the pesticide. For example, the rapid elimination of endrin by one organism and accumulation by another may cause erroneous indications of continuing pollution in a closed system (Fig. 5.1).

Resistance to endrin was found in freshwater shrimp (*Palaemonetes kadiakensis*) (Naqvi and Ferguson, 1970). Shrimp populations gathered from different areas had endrin tolerances that correlated with the amount of endrin in the source water. The 24-hr LD_{50} values for shrimp from a nonpolluted lake, a lake containing cotton-field runoff, a pond draining a cotton field, and a drainage ditch located in cotton fields were 0.9, 2.8, 4.7, and 13.7 ppb endrin, respectively (Naqvi and Ferguson, 1970).

Another source of variation in endrin toxicity is the use of static (with continuously renewed concentration) or continuous-flow bioassay tanks. Endrin levels causing death to the stone-fly naid *P. californica* in static bioassays produced only tremors and convulsions in continuous-flow aquaria (Jensen and Gaufin, 1966). Static conditions may cause oxygen depletion, a buildup of metabolic products, or other conditions detrimental to stone flies.

Following the major fish kill in the lower Mississippi River in 1963, endrin application was reduced, in turn decreasing endrin concentrations in the water (Fig. 7.8) and the wildlife (Rowe et al., 1971).

For example, oysters (*Crassostrea virginica*) from estuaries below New Orleans in 1965–66 had endrin concentrations up to 70 ppb, but the levels were below 2.5 ppb (median 1 ppb) by 1968–69 (Rowe et al., 1971). Adsorption of endrin by sediment would allow endrin to be released back to solution as water concentrations fell, thereby extending the contact time with oysters and resulting in the cycling of the low concentrations of endrin.

The highest endrin concentrations reported were below 100 ppb in 1969, occurring in Pacific oysters (*Crassostrea gigas*) and Atlantic clams (*Corbicula fluminea*) from San Francisco Bay (Modin, 1969). Major rivers draining the agricultural basins of California terminate in the bay, so it appears that endrin runoff was diluted by tidal flow.

Samples of oysters along the Gulf and southeast Atlantic coast in 1966 contained endrin levels up to 70 ppb (Bugg et al., 1967). Negative results were obtained from pelecypods in Long Island (New York) estuaries (Foehrenback et al., 1971) and near Baton Rouge, Louisiana (Novak and Rao, 1965). Data are absent on endrin levels in aquatic invertebrates outside the United States and in more recent (1973 to 1975) years.

8.2.3.4 Fish. The level of endrin accumulated by fish depends upon the concentration and exposure time (Sect. 5.2.2). Largemouth bass and tui chubs rapidly adjust their endrin residues to environmental concentrations (Godsil and Johnson, 1968). Maximum concentration in tui chubs was 198 ppb (1980 times maximum water concentration) during the agricultural growing season when endrin application rose. At the end of the season, as the concentration in water fell to 0.007 ppb, the biota had levels of 4.0 ppb. The largemouth bass showed similar fluctuations (Fig. 8.8).

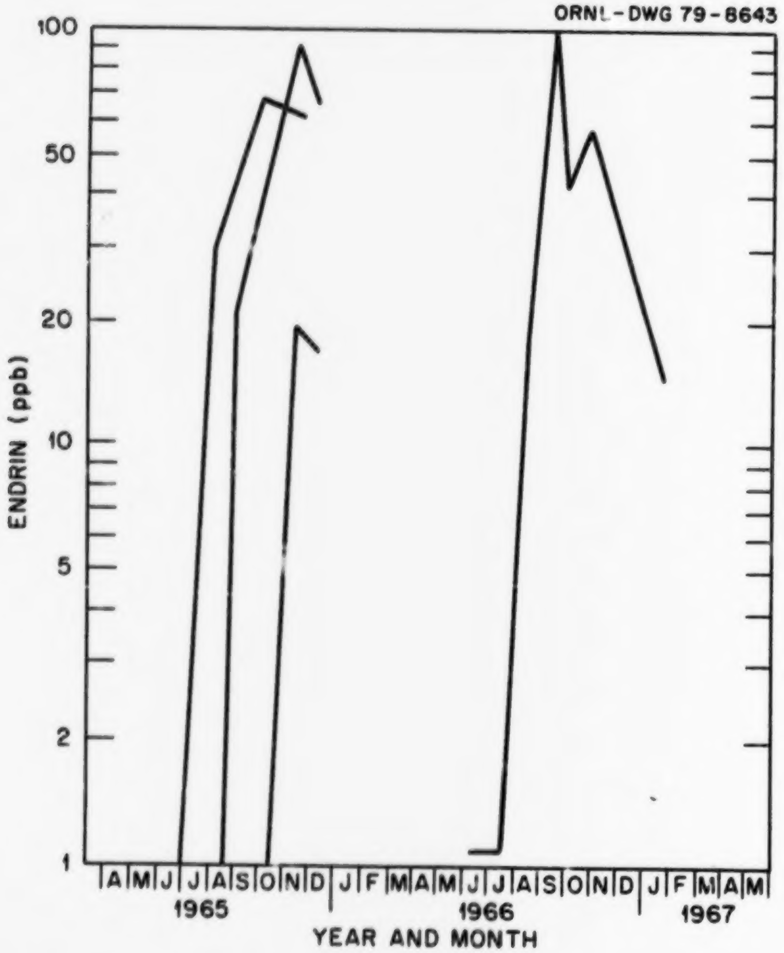

Fig. 8.8. Endrin accumulation in largemouth bass. Source: Godsil and Johnson, 1968.

In general, increasing exposure time and temperature increases endrin toxicity (Lowe, 1965; Pimental, 1971; Iyatomi et al. 1958). A good example of the exposure time and toxicity relationship is demonstrated by the Indian catfish (*Heteropneustes fossilis*) (Saxena and Aggarwall, 1970). Endrin levels of 120, 14, and 5.98 ppb caused 100% mortality in 5, 12 to 16, and 24 hr, respectively, while a level of 5.78 ppb could be tolerated indefinitely. The salmonids appear to experience maximum toxicity at 72 hr, after which increasing exposure times do not cause increasing lethality (Katz, 1961).

Rainbow trout (*Salmo gairdneri*) and bluegills (*Lepomis macrochirus*) show increased susceptibility to endrin with increased temperature (Table 8.9) (Holden, 1973; Katz and Chadwick, 1961; Macek et al., 1969; Pimentel, 1971). Marine three-spined sticklebacks (*Gasterosteus aculeatus*) also have a direct correlation between temperature and endrin toxicity (Katz and Chadwick, 1961). Minor (no values given) temperature fluctuations do not affect endrin toxicity to the Indian catfish (Saxena and Aggarwall, 1970). Mummichogs (*Fundulus heteroclitus*) are most sensitive to endrin in the 20 to 25°C range and do not appear to be affected outside that temperature spread (Eisler, 1970b) (Fig. 8.9). The temperature-toxicity relationships may reflect enzymatic activity (Macek et al., 1969), which generally increases with temperature to an optimum within the tolerance range of the organism. At 30°C the optimal enzyme temperature for the mummichog would appear to be exceeded and toxicity drops.

Table 8.9. Temperature-toxicity relationships for endrin in rainbow trout and bluegills

Species, TL_{50}	(ppb) 1.6°C	 7.2°C	 12.7°C	Relative increase in susceptibility
Rainbow trout				
24 hr TL_{50}	15	5.3	2.8	5.35
96 hr TL_{50}	2.5	1.4	1.1	2.27
	12.7°C	18.3°C	23.8°C	
Bluegill				
24 hr TL_{50}	2.8	1.5	0.8	3.5
96 hr TL_{50}	0.61	0.41	0.37	1.64

Note: TL is median tolerated limit of toxin in aquatic media.

Source: K. J. Macek, C. Hutchinson, and O. B. Cope, *Bull. Environ. Contam. Toxicol.* 4(3): 174-183 (1969). Copyright 1969 Springer-Verlag.

Salinity does not exert much effect on endrin toxicity (Eisler, 1970b; Katz, 1961; Katz and Chadwick, 1961; Lowe, 1965). Interestingly, only estuarine fish (mummichogs) responded consistently to increasing salinity, showing a 20% increase in mortality for a doubling of salinity [from 18 to 36 parts per thousand (Eisler, 1970b)].

Direct application of laboratory LC_{50} to field exposures may not be possible. It appears that acute toxicity generally occurs at higher

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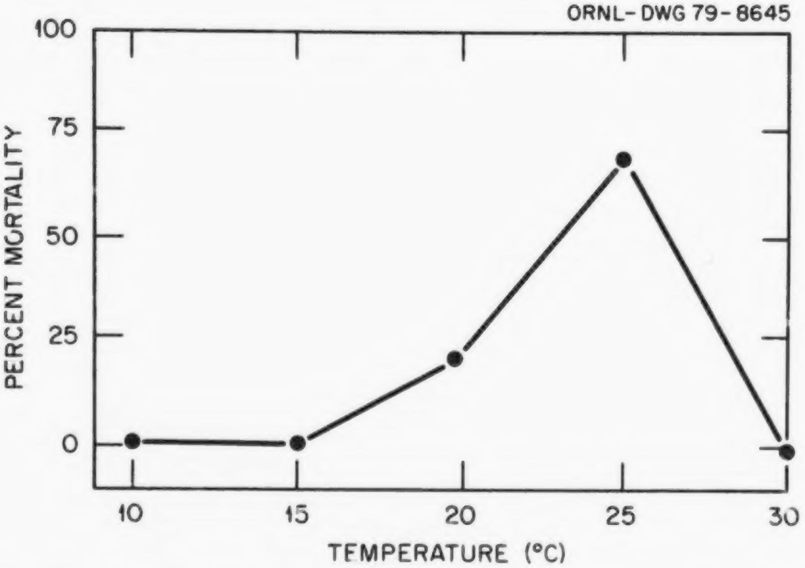

Fig. 8.9. Effect of temperature on toxicity of endrin (0.6 ppb) to mummichogs. Source: modified from Eisler, 1970b.

concentrations in the field than in the laboratory (Johnson, 1968). For example, 40 ppb did not result in a total fish kill in a pond but was well above the laboratory LC_{50} (Johnson, 1968). On the other hand, sublethal field concentrations of as little as 0.009 ppb have produced fish kills.

Static and continuous-flux conditions in the laboratory produced conflicting evidence as to the effect on endrin toxicity (Earnest and Benville, 1972; Holden, 1973; Lincer et al., 1970). However, flux tests are probably more meaningful, since stress may be caused in static water by oxygen depletion and waste buildup (Korn and Earnest, 1974).

Seasonal variation in susceptibility to endrin has been reported by Fabacher and Chambers (1971). Mosquito fish are more susceptible to endrin in the winter and early spring, apparently correlated with low body lipid at those times.

Symptoms of endrin poisoning in the field were similar to those observed in the laboratory (Sect. 5.2.2). In addition, channel catfish from the Mississippi River also had distended abdomens at death (Holden, 1973). Survivors of an accidental endrin poisoning that killed several thousand brook trout and juvenile Atlantic salmon on Prince Edward Island, Nova Scotia, demonstrated unusual downstream movements (Nielsen, 1971).

Fish are among the many organisms that demonstrate resistance to endrin. Maintenance of resistance over several generations in an endrin-free environment suggests a genetic base for the tolerance (Ferguson, 1967).

Endrin-resistant populations may develop by natural selection favoring the rare individual(s) in a wild population that exhibits resistance, and then passing on the genetic capability for tolerance to succeeding generations. Artificial selection in the laboratory when mosquito fish were exposed to endrin for consecutive generations resulted in strains with increased tolerances (Ferguson, 1967). A comparison of fish gathered from uncontaminated areas with populations exposed to heavy concentrations from adjoining cotton fields disclosed as much as a 200-fold resistance in the exposed fish (Pimentel, 1971); the data are presented in Table 8.10.

Table 8.10. 36-hr LC_{50} values for field fish from endrin-polluted and nonpolluted areas in Mississippi

Species	36-hr LC_{50} (ppb)	
	Nonpolluted	Endrinpolluted
Golden shiners	3.0	310.0
Bluegills	1.5	300.0
Green sunfish	3.4	160.0
Mosquito fish	1.0	120.0
Black bullhead	0.37	2.5

Source: data from Pimentel, 1971.

Endrin resistance in fish has the advantage not only of affording protection against endrin but also of providing cross-resistance to other organochlorine insecticides such as dieldrin and toxaphene (Ferguson, 1967). However, resistant fish can tolerate higher body burdens of endrin than do susceptible fish, and it is these residues that present a source of environmental concern. Field-collected resistant mosquito fish contained residues up to 11.95 ppm (Ferguson et al., 1966), while susceptible fish contained up to 0.88 ppm. Resistant fish exposed to 500 ppb for two weeks in the laboratory accumulated endrin in amounts ranging from 201.6 to 214.3 ppm. These high body burdens may adversely affect aquatic communities in two ways: through ingestion by predators and by release to the ambient water.

Endrin-resistant mosquito fish (average residues of 180 ppm) were fed to endrin-resistant and susceptible green sunfish (Finley et al., 1970). The resistant sunfish survived 96 hr, while the susceptible fish died within an average of 11 hr. Ninety percent of the susceptible sunfish regurgitated the mosquito fish but died nevertheless, suggesting absorption of endrin from water. It was found that endrin-treated resistant mosquito fish (exposed to 2 ppm for seven days) contained sufficient endrin to kill predators several hundred times their own weight (Table 8.11). While endrin levels of 2 ppm are not normally encountered under natural conditions, the possibility exists of isolated areas where run-off, equipment washing, or direct spraying may drastically raise endrin concentrations.

Table 8.11. Effect of force-feeding on endrin-resistant mosquito fish (average 890 ppm endrin) to a predator

Predator	Mortality (%)	Time (hr)	Ratio of predator:prey (mean weight)
Red-eared turtle (*Pseudemys scripta elegans*)	72	112.8	757:1
Yellow-bellied water snake (*Natrix erthrogaster flavigaster*)	100	65.4	402:1
Diamond-backed water snake (*Natrix rhombifera*)	100	54.0	714:1
Cottonmouth (*Ancistrodon piscivorus*)	91	27.1	311:1
Bullfrog (*Rana catesbeiana*)	100	15.6	418:1
Redfin pickerel (*Esox americanus vermiculatus*)	100	7.1	35:1
Largemouth bass (*Micropterus salmoides*)	100	12.6	168:1
Bluegill sunfish (*Lepomis macrochirus*)	100	9.4	113:1
Purple grackle (*Quiscalus quiscula*)	100	8.2	77:1
Starling (*Sturnus vulgaris*)	100	1.0	93:1
Coturnix quail (*Corturnix corturnix japonicum*)	100	1.3	134:1

Source: P. Rosato and D. E. Ferguson, *Bioscience* 18(8): 783-784 (1968). Copyright 1968 American Institute of Biological Sciences.

An experiment involving resistant fish in a field environment was conducted by Ferguson (1967). Thirty-nine mosquito fish (each containing approximately 400 ppm endrin) were added to a woodland pool previously stocked with 41 susceptible sunfish. In 16.5 hr, 11 of the sunfish were dead, and only 8 sunfish and no mosquito fish were recovered after eight days. The results indicate that one possible consequence of bioconcentration is the extinction of top piscivores, an occurrence illustrated by the absence of largemouth bass in waters heavily contaminated with endrin (Ferguson, 1967).

Laboratory experiments demonstrate the hazard that resistant fish pose to other fish by means of endrin release to the water. Ferguson et al. (1966) exposed a single resistant mosquito fish to 1000 ppb endrin for 36 hr and then transferred it to 10 liters of tap water. Sufficient endrin was released to kill five susceptible mosquito fish in 38.5 hr, implying water concentrations of 2 to 3 ppb according to bioassay fish tolerances.

Higher trophic levels are not necessarily more resistant than lower ones. And not all fish have the same potential for developing resistance. For example, black bullheads are able to resist only a sevenfold increase in endrin (2.5 vs 0.37 ppb), while yellow bullheads can develop tolerance for a 60-fold increase (75 vs 1.25 ppb) (Ferguson and Bingham, 1966b).

While the benefits of endrin are evidence in the control of insects, the positive use of endrin in fisheries is often overlooked. Sreenivasan and Natarajan (1962) reported on the successful use of endrin in fishery management in India. Carp failed to reproduce in Ooty Lake because mosquito fish, inadvertently introduced, interfered with carp breeding. Prior to treating the lake, test ponds (1 m deep) were sprayed with 15 ppb endrin (no quantity given) twice in one month. All of the mosquito fish were killed. A month later, adult mirror and scale carp successfully spawned in the ponds, and fry and fingerlings were recovered. The shallow margins of Ooty Lake were then sprayed with 20% endrin (4.5 liters, but no area was given). Approximately 90% of the mosquito fish and some adult carp died. However, ten weeks after treatment, carp spawned successfully. The breeding produced many fingerlings, allowing over 53,000 to be taken for stocking other lakes.

In 1967 and 1968, fish from approximately one-half of the sampling stations in the United States had endrin residues ranging from 0.01 to 1.5 ppm, but consistent residues were found at only three stations in cotton areas: the White River, Mississippi River, and Arkansas River (Henderson et al., 1969). While variations occurred, bottom fish and fish from agricultural drainage areas generally had the highest residues. By 1969 no endrin residues were detected in fish from 50 nationwide monitoring stations operated by the Bureau of Sport Fisheries and Wildlife (Henderson et al., 1971).

Endrin residues in catfish from commercial catfish farms were directly correlated to the surrounding number of acres planted in cotton (Crockett et al., 1975; Hawthorne et al., 1974). In 1970, catfish accumulations averaged 0.06 ppm (0 to 0.41 ppm) and were detected in 76% of the catfish (Hawthorne et al., 1974; Crockett et al., 1975). It appears that aerial transport of endrin was the primary source, since sediment, feed, and water were not contaminated (Crockett et al., 1975).

No significant amount of endrin (no level given) was found in commercially caught fish in Canada in 1970 (Reinke et al., 1972). However, endrin concentrations in the waters around Ontario, Canada, increased from less than 3 ppb in 1970 to 1 ppm in 1971 (Miles and Harris, 1971, 1973). Fish, however, remained at low residue levels. Rock bass and bluegills had less than 10 ppb, except in agricultural regions in 1971 (30 ppb), and suckers and chubs ranged from 10 to 18 ppb in 1970, although endrin was not detected in the mud or water.

Worldwide reports indicate that endrin is not a significant pollutant. No residues were found in threadtail, black-tip shark, or catfish from undeveloped or developed areas of northern Australia (Best, 1973). Fish (*Talapia grahami*) from Lake Nakuru in Kenya, an area of intensive agriculture and pesticide use, had low endrin levels of 2 ppb (Koeman et al., 1972). No endrin was detected in a nonmigrating marine bathydemersal (deep-bottom) fish (*Antimora rostrata*) by Meith-Avcin et al. (1973).

8.2.3.5 Effects on community structure. Some examples of the more drastic environmental impacts caused by endrin are worth discussing. Probably the most far-reaching and extensive occurrences were the fish kills in the drainage basins of the Mississippi River.

Beginning in November 1960 and continuing for four years, large numbers of dying fish and shrimp were observed in the lower Mississippi River and adjacent Gulf of Mexico (Anonymous, 1964; Mount and Putnicki, 1966). Endrin was identified as the toxicant by laboratory investigations using extracts from the dead fish. Other pesticides were also found, but feeding different fractions to fish showed the endrin fraction to be the active one. As further support for endrin causation of the fish kills, lethal threshold concentrations of endrin in catfish blood (0.3 ppm) determined in the laboratory corresponded to those found in dying specimens (0.40 ppm or more) from the kills. Catfish and bream from the fish-kill areas consistently had endrin residues of 10 ppb from July 1964 to June 1965 (Novak and Rao, 1965). At the time of the 1963 Mississippi River fish kills, Johnson (1968) reports fish (species not given) residues of 7 ppm in surface waters of 0.1 ppb, and mosquito fish had levels of 6.8 to 11.95 ppm.

Endrin concentrations found in the Mississippi River during fish kills ranged from 0.1 to 0.2 ppb, levels acutely toxic to channel catfish, largemouth buffalo, and gizzard shad (Mount and Putnicki, 1966). Lauer et al. (1966) measured endrin levels of 0.009 to 0.040 ppb in Louisiana streams at fish-kill sites, but later apparently concurred with the data of Mount and Putnicki (1966). In 1964, there were no large fish kills, but all water samples from the lower Mississippi River region were positive and ranged as high as 0.145 ppb (Breidenbach et al., 1967). While fish kills were associated with the first precipitation and runoff following endrin application, endrin residuals persist in soil from one agricultural season to the next and in river sediment. This persistence along with continued endrin usage probably accounts for the continuing presence of endrin.

In another example of an environmental calamity, over 2000 lb of carp and buffalo fish was killed by endrin runoff (U.S. HEW, 1969). Endrin was sprayed on tobacco plants (no concentration or location was given) on a hill above some lakes. A 4-in. rainfall occurred within a few hours of application, flushing endrin into the lakes and causing the fish kill.

A deliberate disaster was created by dumping 0.5 gal of endrin and strychnine into 3-acre Shawnee Lake, Portsmouth, Ohio (Stoltenberg, 1972). Within 24 hr, 4000 fish and other aquatic life were dead. Endrin was found to be stratified in the upper 5 ft of the lake, reaching concentrations of 15 ppb. The toxic effects lasted for at least eight weeks.

To interpret the effects of endrin on an entire aquatic ecosystem it is necessary to understand not only changes in a species but how the changes intermesh and affect the whole ecosystem (Fig. 8.10). In some instances, low concentrations of endrin may produce more severe results than higher concentrations. For example, low endrin exposures may cause resistance in fish, with the corresponding increase in endrin body burdens. Predators on the resistant fish may be killed by ingesting the high endrin level, or they may further transfer endrin across trophic levels as they, in turn, become prey. At high endrin concentrations, all of the exposed fish may be killed immediately, limiting the effects of endrin to mortality in the initial area of contamination.

Endrin may alter the population of an area by differential impact on the various species and their developmental steps. For example, eggs are generally resistant to endrin, so that levels killing adult predators

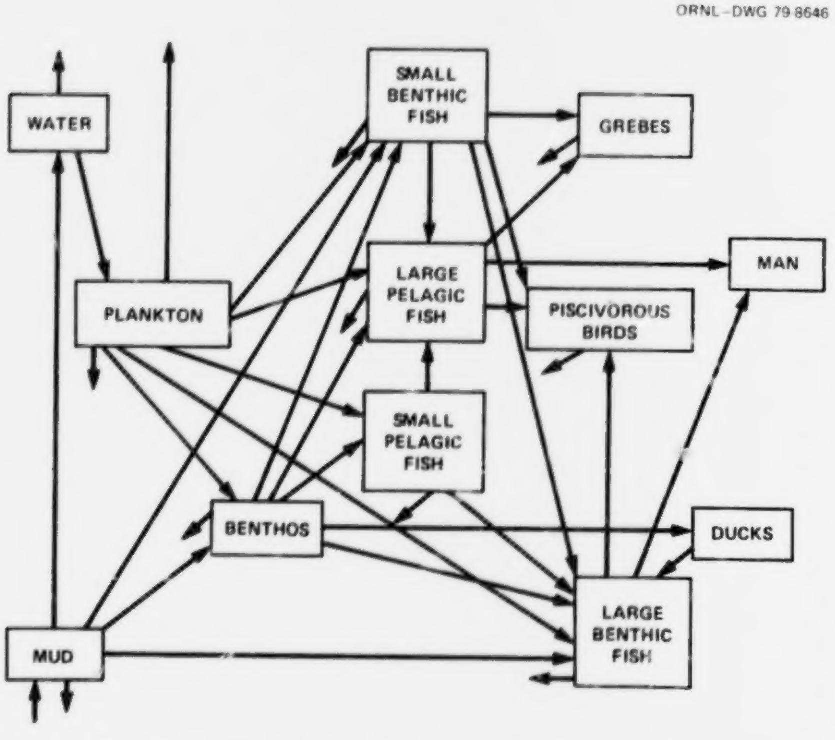

Fig. 8.10. Compartment model of an aquatic ecosystem. Source: R. B. Craig and F. L. Rudd, *Survival in Toxic Environments* (1974). Copyright 1974 Academic Press.

may not affect later hatched populations of prey. Food chains may also be disrupted by the higher toxicity of endrin to crustaceans, insects, and fish than to flatworms, snails, and annelids (Johnson, 1968). Bottom and plankton populations may be depleted without an immediate effect on fish populations, but in time the situation may change.

As Sect. 5 illustrates, a quantitative report on the processes and parameters involved in endrin cycling is impossible, not only because of biological and chemical inconsistencies but also because a completely definitive description of an aquatic ecosystem defies compilation. As a result, partial system studies have been undertaken. A short food chain is studied in order to gain information for comparison with other laboratory or field studies. While model ecosystems have limited applicability to natural conditions, they do provide a basis for comprehensive and prediction of endrin behavior in the aquatic environment.

Metcalf et al. (1973) set up a model ecosystem in an aquarium that consisted of a sloping terrestrial portion interfacing with sand at the water portion. Radiocarbon-labeled endrin (5 mg, equivalent to 1.1 kg/ha) was applied to sorghum seedlings on the terrestrial portion. Fourth instar salt-marsh caterpillars (*Estigmene acrea*) fed until all the leaves were eaten and the fecal products and larvae themselves contaminated the aquatic portion of the ecosystem. The distribution of the radiolabeled products was determined in the ecosystem after 33 days. Several members of the food chain were analyzed: alga (*Oedogonium cardiacum*), snail (*Physa sp.*), water flea (*Daphnia magna*), mosquito larvae (*Culex pipiens quinquefasciatus*), and fish (*Gambusia affinis*); the data are given in Table 8.12. Endrin was highly toxic to the salt-marsh caterpillar, which had difficulty ingesting it. When the water concentration of endrin was 0.06 ppm, both water fleas and mosquito larvae were killed and had to be reintroduced repeatedly. The water was also extremely toxic to the fish, which died after violent convulsions or within a few minutes of being added. The water toxicity persisted for more than 60 days from the initial addition of endrin to the seedlings and apparently occurred at concentrations from 1 to 2 ppb.

The model ecosystem data disclose several important features of endrin transfer in the food chain. The relative stability of endrin is demonstrated by the movement of toxic amounts of unchanged endrin from seedling to caterpillar to aqueous solution. Disruption of trophic levels was shown by the greater sensitivity of primary consumers (water fleas and mosquito larvae) than by primary producers (algae). The secondary consumers (fish) were also sensitive to endrin in solution, dying even before consuming endrin-contaminated food. Further adverse effects from the introduction of endrin were apparent in the high bioconcentration of endrin by snails, resulting in an almost certain lethal dose of endrin to predators.

Analysis of endrin contamination of a natural ecosystem provided results consistent with those obtained in the laboratory. Bingham (1970) compared an endrin-polluted lake (Wolf Lake) with a comparatively clean lake (Mossy Lake) in the Mississippi delta region in 1967 and 1968.

Table 8.12. Distribution of endrin in some components of a model ecosystem

Component	Total $^{14}C^{a}$ (ppm)	Endrin		B.I.[b]	E.M.[c]
		ppm	% total ^{14}C		
Water	0.0134	0.00254	19.0		
Alga	13.62	11.56	84.9	Not given	4,551
Snail	150.58	125.00	82.8	0.0127	49,213
Fish	4.48	3.40	75.8	0.009	1,335

[a] Introduced as ^{14}C-endrin.

[b] Biodegradability index = $\frac{\text{polar products}}{\text{nonpolar products}}$.

[c] Ecological magnification = $\frac{\text{endrin in organism}}{\text{endrin in water}}$.

Source: R. L. Metcalf et al., *Environ. Health Perspec.* 4: 35-43 (1973). Copyright 1973 National Institute of Health Sciences.

However, insecticides such as DDT and toxaphene were found along with endrin in Wolf Lake, so the results probably represent the composite influence. Wolf Lake was chosen for study as being representative of declining productivity as manifested by poor fishing, while Mossy Lake displayed excellent fishing.

Determinations of total insecticide concentration in the two lakes showed that Wolf Lake samples were higher than those of Mossy Lake on every occasion except two, in both of which abnormally high insecticide levels were obtained at the mouth of a cotton-field drainage ditch entering Mossy Lake. Bioassays using bluegills from Wolf and Mossy lakes showed endrin LD_{50} values of 300 and 15 ppb, respectively, a reflection of previous endrin exposure levels. No largemouth bass were found in Wolf Lake, and none survived a stock attempt. Benthic sampling of Wolf and Mossy lakes showed the average number of organisms per square foot to be 37.2 and 54.6 respectively. The composition of the bottom communities also disclosed different characteristics, with small mussels and oligochaetes comprising 8.9 and 54.5%, respectively, of the benthic fauna in Wolf Lake and 56.6 and 0.06%, respectively, of Mossy Lake fauna. Plankton ratios in Mossy Lake compared with Wolf Lake were: zooplankton, 0.84 in 1967 and 2.34 in 1968; and phytoplankton, 1.47 in 1967 and 2.27 in 1968. There was a decrease in both plankton in Wolf Lake relative to Mossy Lake, but no data were given as to whether decreases occurred in Wolf Lake or increases occurred in Mossy Lake. However, taking pollution into consideration, it seems reasonable to assume a decrease in populations in Wolf Lake. If 1967 is taken as a base year, zooplankton have decreased approximately twice as much as the phytoplankton. Since successively fewer numbers of organisms make up increasingly higher food-chain levels (Fig. 8.3), a decreased producer population would be expected to alter the consumer level by at least double. Further support for a threefold decrease in Wolf Lake, rather than the corresponding increase in Mossy Lake, comes from finding that primary consumers in the model ecosystem are much more sensitive to endrin than are primary producers.

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9. ENVIRONMENTAL ASSESSMENT

9.1 PRODUCTION, CONSUMPTION, AND USES

9.1.1 Production and Consumption

Endrin was introduced into the United States in 1951 by the Hyman Company. It is being produced in the United States by Velsicol Chemical Corporation, while Shell Nederland Chemie manufactures endrin in the Netherlands. Endrin is snythesized by the oxidation of isodrin at temperatures below 100°C.

Consumption of endrin is apparently declining. Current consumption figures are not available; however, the figures for 1971, the most recent year for which data are available, indicated that usage had decreased by 35% from the 1964 figure of 984,726 kg (Eichers et al., 1968: U.S. Department of Agriculture, 1968; Andrilenas, 1974).

9.1.2 Uses

Endrin is used as an avicide, rodenticide, and insecticide, the last being the most important. As an insecticide its major application is to cotton. A wide variety of other crops may also be protected from insect pests by endrin. Endrin is also approved for use in the protection of forest seeds against birds, mice, and chipmunks, and for the control of birds on buildings and mice in orchards.

9.2 BIOLOGICAL EFFECTS IN THE ENVIRONMENT

9.2.1 Microorganisms

Only a few microorganisms are able to degrade endrin; but these are also able to degrade dieldrin. Metabolic pathways for microbial degradation are still speculative, but degradation appears to begin on the nonchlorinated epoxy ring, thus forming ketones and aldehydes which may undergo dechlorination. Ketoendrin is the only metabolite positively identified to date. Detoxification does not always result from the degradation of endrin. Metabolites formed may be more toxic and often are more stable. Transformation of endrin by soil microorganisms is increased by anaerobic conditions and highly organic soils. In the ocean, algae are active in endrin transformations.

Endrin does not appear to be excessively inhibitory to ammonifying organisms or decomposers that liberate ammonia. Eno and Everett (1958) concluded that higher plants exhibited signs of phytotoxicity before microbial responses were observed. Many of the soil fungi (including Streptomyces and Penicillia) and bacteria are not affected by concentrations below 1000 ppm. Some algae concentrate endrin manyfold, and growth, respiration (Pfister, 1972; Bollen and Tu, 1971), and photosynthesis may be inhibited to some degree.

Only 2 to 17% of isolated soil microorganisms are active in endrin degradation (Matsumura et al., 1971; Brooks, 1974). Its persistence in the soil for longer than nine months implies continued use will build up toxic levels to nontarget organisms.

Endrin is very persistent under both aerobic and anaerobic conditions, but the creation of anaerobic conditions generally stimulates microbial degradation. Temperature does not seem to affect the degradation of endrin (Pfister, 1972), nor does soil mixing, but degradation is enhanced by the amount of organic material in flooded or naturally occurring soils (Guenzi et al., 1971; Castro and Yoshida, 1971).

9.2.2 Vascular Plants

Endrin uptake by vascular plants has been demonstrated. Plants grown in soil that contains endrin absorb it through their root systems. Endrin is taken up by plant leaves both upon topical application and by absorption of endrin vapors which volatilize from the soil. Endrin uptake by plants from soil depends on the plant, the type of soil, and the endrin concentration in the soil. Residues are usually highest in plants grown in sandy loam and somewhat lower in clay or loam soils. Endrin uptake by the plant is retarded by the presence of silt in the soil.

The absorbed endrin is translocated through the plant. Concentrations vary in the different parts of the plant, decreasing with distance from the site of uptake. As many as five metabolites have been detected in plants after absorption. The rate of metabolism by plants varies with the plant species and probably with mode of endrin application. Endrin and its metabolites are excreted into soil by plant roots.

Endrin is somewhat phytotoxic to vascular plants; however, some adverse effects attributed to endrin are actually caused by the solvent used in the formulation. A 0.03% endrin emulsion produced scorching of the foliage of three varieties of cucurbits after seven days. A 0.04% emulsion produced high phytotoxicity after three days. Endrin can affect the rate of seed germination as well as mitotic and meiotic processes.

The presence of endrin in the soil affects the uptake of various macro- and microelements, either increasing or decreasing uptake, depending upon the particular element, plant species, and the endrin concentration.

9.2.3 Aquatic and Higher Animals

Animals are exposed to endrin through their contact with several components of their environment, including food, water, air, and soil. Endrin may be absorbed through the digestive tract and/or body surfaces.

After an initial rapid uptake, endrin accumulation reaches an equilibrium between elimination and uptake. Both rate of uptake and equilibrium concentration are directly related to the exposure level.

Endrin is not stored in tissues, but remains in a dynamic state, equilibrating with ambient levels. After reaching the equilibrium concentration, the duration of exposure does not affect the amount of residue. However, after chronic exposures, many insects, shrimp, and fish develop a tolerance to increased endrin doses. If the exposure level is lowered, endrin is eliminated from birds, mammals, and aquatic animals.

Aquatic mollusks and arthropods are exposed to endrin in the water, sediment, and food sources. These aquatic invertebrates act as extractors, continually exchanging residues between body fluids and water until reaching an equilibrium. This equilibrium will depend on the concentration of endrin in the water, species, bottom concentration, and exposure time. Endrin is rapidly accumulated initially by mollusks and arthropods, achieving bioconcentration factors up to 49,000 and 2600 respectively (Metcalf et al., 1973).

Endrin is more toxic to aquatic arthropods than to mollusks. Lethal concentrations are less than 10 ppb for several species of marine shrimp, while clams and mud snails survive at levels a thousand times greater, that is, 10 ppm. Sublethal endrin concentrations also affect growth and reproduction of many aquatic invertebrates. Oyster larvae exposed to sublethal endrin concentrations were generally more sensitive than eggs (Davis, 1961).

Direct sources of endrin to fish include food, water, and sediment. Uptake in fish is based on absorption and solubility differences between fat and water, with fish acting as a multiphase exchange system with successive and reversible partitionings between water, blood, and fat. Endrin concentration in water determines the initial uptake rate and equilibrium levels in fish. The uptake from water far exceeds that from food, bioconcentration from water being 2000 times higher than that from food in channel catfish (Argyl et al., 1973). The primary means of endrin entry into the fish body is absorption through gill surfaces.

Endrin is most toxic of the organochlorine insecticides to fish (Eisler, 1970a, b, c). Many factors influence the toxicity of endrin to fish; among these are exposure time, temperature, salinity, body size, previous exposure, flowing or static bioassay systems, species, synergism with other compounds, route of entry, developmental stage, and season.

Mortality occurs in aquatic invertebrates and fish at endrin concentrations as low as 1 ppb in water, while birds and mammals, acquiring endrin from feed sources, exhibit mortality at doses of 2 to 20 ppm. Lower concentrations may cause pathological and reproductive changes, suggesting that chronic exposure to lower concentrations may affect nerve function and metabolism.

The central nervous system apparently is the primary site of endrin toxicity in all animals. The signs of endrin poisoning reflect nerve stimulation, with reactions to acute levels generally following the pattern of hypersensitivity, hyperactivity, tremors, equilibrium loss, convulsions, hyperventilation, and paralysis. Endrin may also inhibit certain enzymes and impair liver function. Chronic endrin exposures of 1 ppm in the diet reduced reproduction of fish and birds by decreasing egg production, and in some cases decreased hatchability and viability of bird eggs were observed.

9.3 EFFECTS ON HUMAN HEALTH

9.3.1 Toxicity

Endrin poisoning in humans has resulted from accidental contamination of foods and has been traced to endrin doses as low as 0.02 mg/kg. Human symptoms of endrin poisoning are convulsions, vomiting, abdominal pain, nausea, dizziness, and headache. In severe cases, convulsive seizures of several minutes duration may be followed by semiconsciousness and can lead to death through respiratory failure. Endrin toxicity appears to be primarily due to its effect on the central nervous system.

Chronic exposure studies in laboratory animals indicate that exposures to low levels of endrin can be toxic. Significant mortality has been observed in mice exposed to 2 ppm endrin in the diet for seven months. Fatalities have been observed following inhalation exposure of animals to 0.36 ppm endrin vapor for 7 hr on each of 130 days.

Humans do not tend to store endrin in significant quantities: residues have not been detected in plasma, adipose tissue, or urine of workers occupationally exposed. Thus despite its high acute toxicity, endrin is relatively nonpersistent in man. Residues are only detected in body tissues of humans immediately after an acute exposure.

9.3.2 Carcinogenesis, Mutagenesis, and Teratogenesis

No malignancies in humans attributed to endrin exposure have been reported in the literature. Endrin can, however, cause mutations in mammalian cells and appears to be teratogenic in mice and hamsters. Endrin exposure may cause an increase in fetal and postnatal mortality, but parental fertility is not affected.

9.3.3 Potential Health Hazards

Human exposure to endrin occurs through the diet via inhalation and by dermal contact. The average dietary intake in the United States from 1965 to 1970 of 0.005 μg of endrin per kg was well below the maximum acceptable daily intake of 0.002 mg of endrin per kg of body weight established by the World Health Organization.

Agricultural workers, home gardeners, and those involved in endrin manufacture and distribution might be exposed through inhalation or dermal exposure. The most significant occupational exposure to endrin comes during spraying of fields, and dermal exposure is almost always greater than respiratory exposure. A threshold limit of 0.1 $\mu g/m^3$ for an 8-hr time-weighted average occupational exposure has been established by OSHA (U.S. Code of Federal Regulations, 1972).

9.4 SOURCES OF PERSISTENCE IN THE ENVIRONMENT

9.4.1 Lithosphere

Direct application is the greatest source of endrin in the lithosphere. Endrin may be applied to the soil directly or indirectly following the treatment of crops. The largest single use of endrin in the United States is for the control of lepidopterous larvae attacking cotton crops in the southeastern and delta states. Endrin-contaminated soils in the United States are located predominantly in the cotton belt or delta states, where endrin is extensively used. Fallout and rainout of airborne particulate matter to which endrin has adsorbed are important mechanisms for contamination of nonagricultural soil or soil on which endrin has never been used. Wind drift and irrigation with contaminated water are also possible routes of endrin to the soil.

Since a large proportion of the endrin entering the environment is initially released to the soil, the fate of endrin in the soil largely determines to what extent the rest of the environment will become contaminated. The persistence of endrin in the soil is dependent upon a wide variety of factors, including soil properties, agricultural processes, topography, and weather conditions.

Endrin is removed from the soil in several ways. Volatilization, leaching into groundwater, wind erosion, and surface runoff translocate endrin to other parts of the environment. Other pathways, such as uptake by plants and ingestion by soil invertebrates, contribute to removal of endrin from the lithosphere. Degradation of endrin through photodecomposition, thermal decomposition, and microbial degradation also occurs, resulting in the dissipation of endrin from the environment. Endrin removal by microbial degradation, however, is contingent upon the presence of an appropriate microbial species and favorable soil conditions. Half-lives ranging from 24 hr to 11.8 years have been observed, depending upon all of the previously mentioned conditions.

Since most of the endrin entering the environment is initially released to the soil, the soil type largely determines to what extent the rest of the environment will become contaminated. Some soil types adsorb endrin, preventing it from escaping to the atmosphere, while others permit extensive volatilization. Climate and topography affect the fate of endrin in the environment also. Rainfall on sloping ground may result in surface erosion, which causes endrin to be transported to the hydrosphere regardless of the soil's adsorption ability.

9.4.2 Hydrosphere

The major source of endrin in rivers and other freshwater bodies is surface runoff from fields and crops following application. Other sources of endrin in the hydrosphere include runoff from endrin-coated seeds, contaminated effluents from pesticide manufacturing and formulating factories, careless aerial application, dumping of unused pesticide into waterways, cleaning spray equipment in rivers and lakes, and contaminated rainout and fallout.

The worst effects of contamination in a given body of water, generally, are recorded immediately following the episode of pollution. Codistillation and sedimentation transfer a large proportion of the endrin elsewhere or render it relatively harmless. However, entire populations of birds or fish may be destroyed during the short period of acute contamination.

Pesticide surveillance of surface waters of the United States river basins has been in effect since 1957. From 1961 through 1964 the number of samples containing endrin progressively increased until, in 1964, 50% of all samples tested were contaminated. However, over this time period, consistent contamination of a large percentage of the samples occurred only in the lower Mississippi basin. Highest concentrations recorded during this six-year period were also from this area. Endrin usage in this area is apparently declining, since the residue levels reported in 1970 and 1975 were only 20 ppt as compared with a maximum of 214 ppt reported in 1963.

The highest concentration of endrin in the sediment of any river basin monitored between 1970 and 1975 was found in the lower Mississippi. This value, 6700 ppt, was significantly less than the amount found in 1964, that is, 10,000 ppt.

Removal of endrin from the hydrosphere occurs by several routes. Volatilization and codistillation reallocate endrin to the atmosphere. Dissipation of endrin from the hydrosphere occurs as a result of biochemical and photochemical degradation. Degradation by aquatic microorganisms has been reported and probably contributes to some extent to the dissipation of endrin from the aquatic environment.

9.4.3 Atmosphere

Vaporization of endrin from treated soils and crops is believed to represent the major source of atmospheric contamination. In one experiment, only 6% of the endrin applied on cabbage plants could be detected on the soil or leaves four weeks after treatment. Codistillation from water, wind transport of pesticide-laden dust, aerial drift from spraying operations, and vapor emissions from industrial sources also contribute to the presence of endrin in the atmosphere.

Since atmospheric monitoring equipment and technology are extremely complex and expensive, they are not in common use. However, the highest endrin concentration in air reported to date was 58.5 mg/m^3 at Stoneville, Mississippi, in 1971. Atmospheric endrin is present both in the vapor phase and adsorbed to wind-borne particulates.

Some dissipation of endrin in the atmosphere via photodecomposition is possible; however, no real evidence that this transformation actually occurs is available. Thus the major hazard involved in the release of endrin to the atmosphere is the subsequent contamination of the hydrosphere and the lithosphere.

Atmospheric transport of endrin far exceeds any other mechanism and is considered to be primarily responsible for the worldwide dissemination of the pesticide. Transport of endrin to the open ocean is thought to occur largely via the atmosphere, from which it is removed by particle fallout or precipitation washout. After deposition in the ocean, endrin may adsorb to the organic materials present and later be revolatilized, or it may adhere to large particulates and be deposited in the sediment. Ocean sediment therefore represents the ultimate sink for endrin and related compounds. The fate of endrin in the ocean sediment remains unknown.

9.4.4 Detection and Analysis

Many techniques for endrin determination have been reported. These include bioassays, total chlorine content, silylation, acetylation, spectrophotometry, and colorimetry. The most sensitive methods available, however, both qualitatively and quantitatively, involve variations in gas chromatography. Gas-liquid chromatography methods are rapid and highly sensitive, permitting detection of a very small quantities of endrin residues.

Although analytical methods are adequate in determining the presence of endrin residues, detection of endrin in the environment is rather difficult at times because of the inadequacy of sampling methods.

9.5 STANDARDS AND REGULATIONS

Several concentrations have been recommended and/or established as acceptable environmental levels for endrin residues. A threshold limit value of 100 μg/m^3 was adopted by the American Conference of Governmental Industrial Hygienists for atmospheric concentrations. While no standards have been set for water concentrations of endrin, 0.1 ppb has been suggested as a maximum reasonable allowance for potable water. Maximum acceptable daily intake for humans was established at 138.2 μg/day by the World Health Organization (1973).

Concentrations of endrin found in air and water are well below accepted limits, except for sporadic high levels in the immediate vicinity of endrin usage. The maximum air concentration of endrin in 1975 was found at Jackson, Mississippi (0.5 mg/m^3). The maximum concentration of endrin in drinking water (23 ppt) was found in Louisiana, from a high endrin usage area, and was less than one-fourth the suggested allowance for potable water. The average daily intake of endrin by man according to the latest available figures was 0.033 μg; this is far below the acceptable daily intake established by the WHO/FAO (1973).

9.6 ENVIRONMENTAL IMPACT

Worldwide monitoring data show that endrin occurs at very low levels. However, endrin has the highest acute toxicity of the common organochlorine insecticides, making even low levels a potential threat to the environment.

Major uses of endrin occurred in the 1960s in the southeastern cotton-producing areas for control of lepidopterous larvae. However, since the large-scale fish kills in the Mississippi River and delta region in the mid-1960s, the levels of endrin in the United States have progressively declined. Environmental contamination is mainly restricted to those areas where intensive use has occurred. Concentrations of endrin, away from areas of heavy use, are generally below the levels of detection.

Bioaccumulation of endrin has been reported in blue-green algae, and growth inhibition occurs in several species of algae and phytoplankton, after endrin exposure. Vascular plants absorb, translocate, and metabolize endrin, but it is generally nontoxic to them. Aquatic invertebrates and fish can bioconcentrate endrin from the water. It is the most toxic to fish of all the commonly used organochlorine insecticides. Birds are extremely susceptible to endrin, with mortality commonly occurring. Mammals rapidly degrade and eliminate endrin even though it can be extremely toxic at times. Endrin in not stored in mammalian tissues, and residues can only be detected immediately after an acute exposure.

The threat to nontarget organisms, mainly birds and fish, does exist becuase of their high degree of susceptibility. However, since endrin usage is declining, the threat for environmental contamination is reduced. The possibility of human food, water, or air supplies being highly contaminated with endrin apparently exists only in those areas immediately adjacent to high endrin use areas.

However, since endrin is such a highly toxic pesticide to fish, birds, and mammals, any increased usage would pose a serious threat to the environment in localized areas.

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16. ABSTRACT

This document is a multidisciplinary review of the health and environmental effects of endrin. Included in the review are a general summary and a comprehensive discussion of the following topics: physical and chemical properties; synthesis and use; analytical methodology; biological aspects in microorganisms, plants, wild and domestic animals, and humans; distribution, mobility, and persistence in the environment; assessment of the present and potential health and environmental hazards; and review of standards and governmental regulations. More than 600 references are cited.

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